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Listerian Oration.[1]

By S. V. SEWELL, M.D.,
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Introduction.

FIRST may I thank the medical profession of this State for the great honour they have done me in naming me the Listerian orator. There is no greater name in medicine than that of the man we are met to honour tonight. Lister has, with Pasteur, conferred greater blessings on mankind than any other individual before or since. The whole subject of cerebral localization would not have advanced beyond the position it had reached with the wonderful clinical and pathological research of Hughlings Jackson had not the work of Lister made aseptic surgery possible. Horsley, Cushing and Foerster could not have attempted their operative research work. Therefore in this, as in almost every other branch of medical and surgical work, we thank God for the gift of Lister.

[1] Read at a meeting of the South Australian Branch of the British Medical Association in June, 1937.

The Evolution of the Cerebral Cortex.

In considering the subject of cerebral localization it seems wise to refer briefly to the evolution of the cerebral cortex from a phylogenetic point of view.

In primitive forms an aggregation of nerve cells appears at the head end of the nervous system. It is the head end which explores the environment, and this is supplied with receptors, the stimuli from which determine reactions of the whole body, whereas stimuli from the extremities determine, for the most part, reactions confined to the particular limb from which they have been conveyed to the central nervous system; hence the increasing

dominance of the head segments. This aggregation of nerve cells, or brain, becomes proportionately bigger and more complex as the environment of the animal becomes more varied and the struggle for existence more intense. In fish, amphibians and lower mammals the sense of smell far transcends the other head receptors in importance, and by the agency of olfactory stimuli the animal searches for its mate and locates its food. In these types the projection area for smell constitutes nearly the whole of the evolving forebrain or cerebral cortex.

In mammals, however, other distance receptors, situated in the retina and internal ear, play an increasing part in bringing the animal into touch with its more complex environment. The cerebral cortex becomes increasingly complex and provides the machinery through which the animal is able to cope with its complex environment, and the animal depends for the perpetuation of its species less on fecundity and more on its ability to react to danger at a distance.

The brain gradually evolves and projection areas for hearing and vision become increasingly represented in the evolving cerebral cortex. By the time carnivora are reached there is a well-marked cerebral cortex with projection areas for vision, hearing, smell, touch and proprioceptive stimuli. For the first time areas adjacent to the projection centres are found to be differentiated, the neurones from them not leaving the cortex, but acting as connecting links between one part and another. So the mechanism for associative memory and inhibition is evolved. One has only to recall the skilled activities of a well-trained gun dog or sheep dog to realize how true this statement is.

When, however, the upright position is assumed, the range of awareness is greatly increased, and the distance receptors, subserving vision and hearing, become of paramount importance, determining as they do, not only the reactions of the whole body to changes in the environment, but also anticipatory reactions to stimuli from the complex environment at a distance. At the same time the sense of smell becomes of comparatively small importance once the head is lifted from the ground with the assumption of the upright position, the increasing environment bringing about a gradual change in the relative size of the projection areas. Thus the olfactory cortex is insignificant in size by the time primates are reached, while the areas for hearing, vision, and bodily sensation are very large. This is not the most striking change that has taken place. For, simultaneously, there have been a great development and differentiation of association areas between and linking up the projection centres, one with another. It is these association areas that determine the great size of the human brain. Posteriorly, the great association area has developed between visual, auditory and general body sensation projection areas. In the human brain this area has become so extensive that the visual sensory cortex has been pushed backwards till it occupies an area almost entirely mesial above and below the calcarine fissure. Peculiar to man, also, is the evolution of a higher association zone, or prefrontal lobe, the function of which will be discussed later.

With this great increase in area of the cerebral cortex there is an enormous increase in cell complexity. In the human cortex there are at least five layers, two supragranular and two infragranular. In types below mammals there is little, if any, differentiation of the supragranular cortex; but, right up through the mammalian scale it is the supragranular cortex which is progressively increasing in depth, and this is due to great aggregation of pyramidal cells, appearing in the second layer of the cortex. This second layer appears first in the projection areas, and in the human brain matures first in these regions. In the adult, however, it reaches its greatest complexity and depth in the posterior associational zone and in the prefrontal area.

There is little variation in the depth of this second layer from individual to individual in the posterior associational zone, but definite differences in its development are noticed in the prefrontal area. In mental deficients it is poorly developed, and in dementia it undergoes degeneration. With its degeneration are correlated loss of judgement and loss of power of concentrated thought with its selection of posterior associational perceptions in accord with the subject under consideration and its disregard of unsuitable ones. In degeneration of the second layer of the cortex in the prefrontal region the individual is a prey to every chance afferent impression, and consecutive thought is greatly interfered with. The posterior zones may be said to be "free-wheeling". Such an individual has greatly diminished emotional and sexual control.

The working of the machine so evolved can be judged only by its motor exhibitions. These may be purely expressive of emotional states or may be complicated voluntary acts, giving evidence of higher cerebral activity.

The movements of emotional expression are primitive and are not carried out by the pyramidal mechanism, but probably by basal ganglia and midbrain arcs, since they are present in earliest infancy, before the myelination of the pyramidal tracts, and persist in adult life in spite of lesions of the pyramidal tracts. However, association fibres freely connect the cortex and basal ganglia. Thus, emotional expression colours all our cerebral processes and often leads indirectly to most complicated truly cerebral activities.

In frontal lesions, emotional expression is largely freed from inhibition and becomes greatly exaggerated.

Speech.

The highest forms of motor expression that have been evolved are the complex motor activities resulting in speech both spoken and written. Speech is first learned by the little child through imitation of sounds heard, which are reproduced as the result of stimuli passing from the auditory projection area to the motor area. This form of speech can be acquired by birds and is not necessarily associated

with any appreciation of the meaning of the words used. A sad illustration of this occurred some years ago at a sanatorium which I have been in the habit of visiting. The patients captured a young magpie and taught it quite a number of phrases, and great was their joy when it spoke to members of the visiting staff, saying: "Oh, George, don't you think you are a gussie?" However, retribution was at hand. In the springtime the lady made advances to numbers of wild males. These left her in terror when she remarked: "Oh, George, don't you think you are a gussie?" She built a nest, laid three eggs and sat on them for an unconscionable time, but, alas, they were sterile. A tragedy of the over-education of the female.

Words are mere sensory impressions unless they are definitely associated with the objects of which they are symbolic. This necessarily brings into the speech mechanism the visual area; hence the importance of visual memorial images in the naming of objects. A further important stimulus to the language mechanism is the orderly registration in the post-central and parietal areas of proprioceptive stimuli from the muscles in action as words are being produced. Later, if or when the child learns to read or write, a further great impetus is given to the language mechanism, and an educated person acquires the greater part of his sensori-memorial images as the result of this further development. Words, arriving at the cerebral cortex, either by the auditory or visual pathways, vary very much in their symbolic value. Prepositions, simple adjectives, may arouse simply a visual or auditory perception of the word itself, while proper names and adverbs will often arouse a whole series of concepts in which the whole mantle of cerebral cortex takes part. The same word seldom arouses twice the same series of conceptions, because innumerable alternative pathways are present, and the immediate connexions made will depend on the condition of the synapses at the moment.

Unfortunately language, once it is acquired, becomes in most people largely automatic, and individuals pass on things that they have heard, or read parrot fashion, without ever attempting to correlate in their own mental processes the words they are using. Thus a great many people seldom think out a proposition throughout their lives. The language mechanism, either when silently used or when used for speaking or writing, is a symbolic mechanism for the integration of thought, and words are its symbols. Accordingly, any form of aphasia must necessarily be associated with some degree of mental impairment. If the lesion is in juxtaposition to the auditory area, jargon speech results, and this only mirrors an equal degree of mental confusion in the individual. However, such a lesion, if unilateral, and particularly if it occurs in childhood, may be gradually and completely compensated for by the activities of the unaffected side. The interference with mental processes is infinitely less in lesions of the intermediate precentral area associated with some verbal aphasia; but in such cases, when careful tests are made, some mental impairment is found. It is only in subcortical anarthria, as opposed to cortical verbal aphasia, that the mental condition is unimpaired.

The brain seems to have an almost infinite capacity for using symbols; extra languages are much more readily acquired by those who can use several, and a wide vocabulary is essential for conciseness of thought. Other symbolic systems, such as those used by the mathematician, the painter and the musician, are somewhat less complicated than the language mechanism.

The Importance of a Full History.

Before discussing in detail the problem of cerebral localization I should like to stress very strongly the importance of getting a complete history of the onset of the condition concerning which you are asked for an opinion. Unfortunately the specialist only too often sees cases, particularly of cerebral tumour, for the first time when a grave degree of increased intracranial pressure has developed. In such cases false localizing signs, due to the indirect effects of the great pressure, are frequently present, and it may be quite impossible by mere examination to disentangle these from the signs produced by abrogation of function due to the actual lesion itself. Again, in such cases the patient is often quite unreliable in his statements, and one has to depend on the statements of others to learn what symptoms and signs were present in the earlier stages of the disease. Such outside information is often woefully inadequate and we are forced to use ventriculography, a somewhat dangerous and often inadequate aid to localization. It is, however, essential that we should make every effort to obtain as full a history as possible of the earliest developments, since the early signs will be due to altered function produced by the lesion itself. I quote one case in illustration.

Two years ago a patient was sent to me from New South Wales. He stated that for six months he had been suffering from attacks beginning with a queer feeling in his left foot followed by working of the left ankle, spreading up to the knee and hip. These attacks were accompanied by a feeling of giddiness and great distress, but no loss of consciousness. However, his wife volunteered the statement that recently he had had two attacks in her presence, beginning in the same way, but followed by unconsciousness, in which the whole of the left side had been simultaneously convulsed and the head and eyes had turned strongly to the left—a typical description of an extrapyramidal attack. She also stated that in the minor attacks her husband only felt the leg working, but no actual movement had been noticed in any attack. This extra information made it quite clear, before a physical examination was made, that the lesion was high up in the parietal lobe, behind the post-central area. Not infrequently such a satisfactory history makes examination almost unnecessary. This case is of further interest in that an active spongioblastoma was removed at operation and that a series of applications of deep X ray treatment to the area followed. The patient remains well, and so far has had no recurrence of symptoms and presents no discriminative sensory signs whatever to physical examination. This is unusual, as spongioblastomata are not usually radio-sensitive.

The Method of Examination.

It has been my practice for many years, when presented with a problem of cerebral localization, to study the case in terms of function of the various cerebral lobes, beginning with the occipital lobe and working forwards. If patients are examined in this systematic way, the beginner in neurology is not likely to overlook any lesion involving the projection areas.

Lesions of the Occipital Lobe.

In lesions of the occipital lobe complete hemianopia or even a complete quadrantic hemianopia is most unlikely to be present if the lesion is cortical, since either necessitates a very extensive involvement of the calcarine area. The most usual finding is a partial upper or lower quadrantic hemianopia, according as the lesion is below or above the calcarine fissure. The positive findings associated with such a lesion are visual Jacksonian epileptic attacks, usually without loss of consciousness and without any organization, since an intact visual cortex is necessary for the organization of visual hallucinations. The hallucinations in these cases usually consist of mere flashes of light in one field or a confused sense of something moving in that field. A complete hemianopia is seldom seen in cerebral cases, except with lesions involving the optic radiation just as it is leaving the posterior limb of the internal capsule before it divides into its two main bundles. Similarly, a complete quadrantic hemianopia is usually associated, if it be in the upper field, with a lesion in the temporal lobe involving the bundle as it winds round the descending horn of the lateral ventricle and passes backwards to terminate in the infracalcarine area. As this bundle is approaching the occipital area, beneath the floor of the lateral ventricle, it gradually fans out and a complete quadrantic lesion becomes less and less likely.

A deep subcortical lesion in the parietal region may involve the other branch of the optic radiation going to the supracalcarine area and so produce a complete inferior quadrantic hemianopia. Such a lesion is almost inevitably associated with characteristic disturbances of bodily sensation, because of its interference with other sensory fibres, which are in juxtaposition. Since the occipital cortex is itself intact in these cases of complete quadrantic hemianopia, if Jacksonian sensory epileptic attacks occur, they are often well organized and most complicated, differing in detail from attack to attack. In making an examination of the occipital lobe, it is well to test the patient's visual memories by asking him to describe something that should be quite familiar to him, such as the arrangement of the furniture in one of the rooms of his house. Despite the fact that the visual representation is completely bilateral, he will often show confusion of visual memories if the lesion is at all an active one. In right-handed people lesions further forward, in the region of the angular gyrus, are always associated with some degree of aphasia and slight mental confusion. If the patient has not learned to read or write, the defect presents itself as a difficulty in naming objects, due to an interference with visual sensori-memorial images; but if he has learnt to read and write, a more marked disturbance of the language mechanism occurs, with loss of the ability to interpret written symbols; in an extensive lesion reading becomes impossible. However, in young people the cortex of the opposite side can gradually compensate for the destroyed area, and aphasic manifestations gradually completely disappear. This is well illustrated by a case to be described later.

Lesions of the Parietal Lobe.

Thanks to the work of Foerster, of Breslau, our knowledge of the symptomatology of lesions of the parietal lobule has been considerably increased. In lesions of the posterior part of this lobule there may be no definite negative findings; but the positive signs, demonstrated by fits arising in this region, are quite definite. An intense sensation of vertigo, associated with general discomfort down the whole of the opposite side of the body, may be the only manifestation of such a fit; but frequently these symptoms are followed by extrapyramidal motor manifestations, in which, almost simultaneously, the head and eyes turn to the side opposite the lesion, the upper limb is abducted and flexed, and the hand is pronated, with the fingers and wrist flexed, the leg being flexed at the hip and knee and dorsiflexed at the ankle or completely extended at all joints. Flail-like movements, flexion and extension may then occur and a similar set of motor manifestations may spread to the opposite side. With these profound attacks unconsciousness is usual. Following such an attack, general diminution of sensation in the side affected by the fit may transitorily be present. Recently Dr. Coates operated on such a patient for me.

This patient was having frequent attacks in which vertigo and painful discomfort down the right side were followed by rigidity in the extensor position of the right arm and leg, and violent tremor, affecting the whole of the right side, followed by flail-like extension and flexion of upper and lower limbs and forced movements of the head and eyes to the right. I had the opportunity of observing a number of these attacks, and the patient remained conscious throughout, and begged to be held because she felt she was falling out of the bed to the right. At the close of the attacks sensation of all kinds was diminished down the right side; but no signs of pyramidal tract involvement could be determined. A large tumour was removed by Dr. Coates. The patient lived a few days only after the operation.

Foerster has shown that stimulation of this portion of the parietal lobe in patients operated on under local anæsthesia, although no lesion of the area was present, reproduced exactly the same clinical picture.

In lesions further forward in the parietal lobule impinging on or involving the postcentral region, the negative findings are often quite definite, in that the discriminative qualities of sensation are involved. If the lesion is behind the motor area for

the arm, there is, in the opposite upper limb, a slowness in appreciation of touch, a defect in localizing power somewhat proportionate to the size of the lesion. Similarly, appreciation of sense of position, particularly of the small joints in the opposite hand, is defective, and with these defects there is inevitably some degree of astereognosis in the hand opposite the lesion. Slight ataxia on voluntary movement of such a limb is to be found. Slighter sensory signs may be present in the opposite lower limb; but, more frequently, no objective findings can be determined. If a lesion be present behind the motor leg area, on the mesial surface of the lobule, similar sensory defects are usually present in the opposite lower limb, the upper limb being unaffected. The positive findings in these cases are characteristic. In contradistinction to the fits characteristically associated with lesions of the posterior part of the parietal lobule the sensory fits follow a definite march. If they begin in the hand they run up the arm, down the trunk, and down the leg, to the foot. In lesions on the mesial surface the fits often begin with a violent desire to micturate, and paræsthesiæ appear in the opposite foot, then running up the leg to the trunk and down the upper limb to the fingers, and then spread to the neck, face and larynx. Frequently these sensory phenomena are followed by a similar march of Jacksonian motor phenomena, at times followed by a generalized convulsion of both sides and profound loss of consciousness. Following such Jacksonian attacks as have been described, without loss of consciousness, the negative findings are temporarily very much increased and, added to those previously described, a transient flaccid palsy, with loss of deep reflexes in the affected limb, may be present. If the lesion be behind the face and arm areas, on the left side, in a right-handed person, there is inevitably some degree of interference with the reception of proprioceptive stimuli from the muscles used in the production of speech which leads to some slight ataxia of the articulatory muscles. The absence of the orderly flow of proprioceptive stimuli from the muscles used in speech inevitably leads to an interference with the flow of language. This does not show itself in short phrases; but, on attempting continuous speech, the patient frequently gets lost and fails to reach his goal.

In contradistinction to most other workers, Foerster has shown that in man the postcentral region responds to electrical stimuli with individual movements in the same way as the Betz cell area itself, and such postcentral stimulation may produce the typical march of a Jacksonian epileptic fit, but always with sensory concomitants. A considerably greater current is necessary to produce such movements from the postcentral region than from the Betz cell area, and such movements cannot be produced on postcentral stimulation if the patient be under the influence of a general anæsthetic. It is this fact which led other observers, such as Horsley, to take the view that the postcentral gyri were unexcitable.

In 1908 I saw Sir Victor Horsley operate on a patient who was suffering from very frequent attacks of Jacksonian epilepsy beginning in the right hand and spreading up the arm. The arm area was carefully delimited by stimulation with a bipolar electrode, and no movements were obtainable from the postcentral region, even on increasing strength of the current. However, this patient was fairly deeply anæsthetized with a general anæsthetic during the whole of the operation. In that case the whole of the area, which on stimulation produced movements of any part of the upper limb, was removed to the full depth of the cortex.

It is clear that these isolated muscle group and single muscle movements produced by stimulation of the postcentral region when the patient is under the influence of a local anæsthetic, are mediated by the corresponding Betz cell area, since Foerster has shown in many cases that after removal of different parts of the Betz cell area stimulation of the postcentral region fails to reproduce these movements; but strong faradic stimulation still produces the characteristic extrapyramidal movements affecting the whole of the opposite side simultaneously.

Time will not allow of a detailed discussion of the well-known positive symptoms associated with irritation of the Betz cell (motor) area itself; but again, Foerster has increased our knowledge of this area by demonstrating in repeated instances that on the mesial surface of the brain, just below the Betz cell area, in the paracentral lobule, there is an area stimulation of which produces micturition and defæcation. If this area is stimulated, involuntary micturition and defæcation immediately occur, usually followed by an attack of Jacksonian epilepsy, beginning in the toes and spreading up the legs, and which may progress up the trunk, down the arm to the fingers, and then to the muscles of the neck and face. My ignorance of this motor area for micturition led to my failing to urge operation on a patient who consulted me some five years ago.

The patient came complaining that for six months he had been suffering from what he called "queer turns". Without warning micturition would suddenly occur, and in three attacks only this act of involuntary micturition was followed by twitching, on two occasions of the toes of the right foot, on one occasion of the toes of the left foot. The attacks were quite transient and were unaccompanied by any mental obscuration. The patient had no headaches and no optic neuritis, and many neurological examinations failed to reveal any abnormality. He remained for a month under observation, during which time he had no attacks. The fact that the attacks varied from side to side led me to the view that there was probably a tumour of the *falx cerebri*. I merely suggested, but certainly did not urge, operation. A year later this patient presented himself with bilateral paresis below the knees and typical bilateral pyramidal tract signs in the lower limbs. Headaches were severe, double optic neuritis was present, and he was having typical Jacksonian epilepsy, frequently beginning with micturition, and a march of convulsive movements beginning with one or both feet, followed by loss of consciousness. At operation Dr. Kilvington attempted to remove a large meningioma of the *falx cerebri* in the region of the paracentral lobule. A great deal of bleeding from the longitudinal sinus interfered with the operation, and the patient died a few days later.

This patient would almost certainly have been saved by operation a year earlier if I had had the knowledge of the area for activating micturition, as

I should under those circumstances certainly have insisted on operation.

Foerster's observations on the negative results of the removal of areas of the Betz cell, or so-called motor cortex, are entirely in accord with those of Horsley and many other surgeons.

In Horsley's case the immediate result of removing the arm area of a patient suffering from Jacksonian epilepsy, beginning in the hand, was a complete flaccid palsy of the upper limb concerned. In a few days, however, reflexes returned and, speaking from memory, as I had the chance of watching this case, within a few weeks some voluntary movement of the large joints appeared. Flexing the elbow was associated simultaneously with abduction of the arm at the shoulder joint, flexion of the wrist, pronation of the forearm and some closure of the hand. Such movements Foerster has now demonstrated to be extrapyramidal in origin. At this stage slight spasticity of the limb was present. Twelve months later I spent a Sunday morning with Sir Victor Horsley photographing this patient in the act of tying his tie and using the affected hand for other semi-skilled movements. However, at that stage he had not yet regained movements of individual fingers; but in flexing the elbow the associated movements of the shoulder, wrist and hand, which had been present six months earlier, had disappeared.

It is probable that in this case the skilled movements which were returning, to some extent at least, were being mediated by the uncrossed fibres from the opposite hand and arm centres.

Foerster reports an illuminating case of bilateral injury of the paracentral lobules, in which bilateral paralysis of the toes and feet was associated with complete loss of control of the bladder and rectum.

Lesions of the Frontal Lobe.

Concerning the intermediate precentral area (Campbell) or "area six" of Broadmann, a great deal of legitimate debate has taken place. Here again the work of Foerster has shown that, except from its upper and anterior portion, stimulation under a local anæsthetic produces individual muscle group movements, exactly similar to those produced by stimulation of various parts of the postcentral region. Again, he has established the fact that these individual movements of the contralateral sides are mediated by way of the precentral or Betz cell area, since they can no longer be produced after extirpation of the corresponding Betz cell area. It is clear from the work of Foerster and Horsley that the opinion of Fulton and his school, who ascribe the spasticity of cortical lesions associated with monoplegia or hemiplegia entirely to associated lesions of the intermediate precentral area, is quite untenable. The few cases of lesions in this area which I have had the opportunity of observing, have shown as their clinical picture some slight degree of hypertonicity affecting the contralateral limb with, if it be the upper limb, a tendency to a group phenomenon (inability to relax quickly the clenched fist at command), and if the lesion be higher up in front of the leg area, a similar inability to relax at command the muscles in action in the lower limb. With these phenomena is associated a considerable degree of interference with the carrying out of skilled movements of the affected part, and typical apraxia results. As S. A. K. Wilson has suggested, verbal aphasia resulting from a lesion of any considerable size in the lower part of this area may well be looked upon as an apraxia of the musculature used in speech. The positive findings associated with lesions in this area are motor Jacksonian fits with a typical march, and quite similar to those produced by irritation of the immediate postcentral area, except that the patients complain of no sensory phenomena during the attack. Physical examination between attacks usually elicits slight increase in deep reflexes on the contralateral side and absence of the superficial abdominal reflexes, which disappear also in precentral or postcentral cortical lesions, since the arc for maintenance is cortical; but the plantar reflex is usually flexor. Foerster has shown that the upper and anterior portion of this region yields to strong stimulation, typical extrapyramidal movements affecting the whole of the contralateral side simultaneously with turning of the head and eyes to the opposite side, quite indistinguishable from the movements produced by stimulation of the parietal lobe. The clinical picture, therefore, associated with lesions of the intermediate precentral area is well defined. It would seem that for the accurate carrying out of skilled movements three areas are essential, the intermediate precentral, the precentral (or Betz cell) area and the immediate postcentral area. These skilled movements are initiated from the frontal region and mediated through the precentral or Betz cell area. It is obviously important, if these movements are to be carried out quickly and accurately and with little effort, that the posture of muscles, at that moment being maintained at the cerebellar level, should be inhibited, and to this end a simultaneous outflow of inhibitory impulses by way of the fronto-pontine tract must be essential. For the continuance of the skilled act the immediate registration of proprioceptive stimuli in the postcentral area from the muscles in action is necessary. Thus these three areas are constantly in action under frontal dominance during all skilled movements. The intermediate precentral area is the dominant partner of the three, since it not only cooperates with the other two in determining the exact outflow to various groups of muscles by the pyramidal tract fibres, but at the same time determines a constantly varying inhibitory action over the cerebellar postural mechanism to facilitate the action of the pyramidal outflow.

A complete lesion of the precentral region itself will necessarily abolish voluntary movements altogether, but extensive lesions of either the intermediate precentral or the postcentral region produce apraxia or ataxia, as the case may be. Two cases are briefly quoted to illustrate the symptom complex of lesions in this region.

One patient, whom I saw in 1912 in consultation with Dr. J. R. Lee, now practising in London, stated that for three months she had suffered from attacks, beginning suddenly, without any sensory symptoms. The eyes and head and also the body turned violently to the right till the patient completely rotated. On several occasions she

had fallen to the ground in the attacks, and the whole right side had been simultaneously convulsed without loss of consciousness. Between attacks she felt quite well and had no headaches. The only physical sign to be found on a complete neurological examination was absence of the right superficial abdominal reflexes. In the absence of any positive or negative sensory signs it was decided to explore the frontal lobe high up in front of the intermediate precentral region. A small hydatid cyst was found and evacuated. The patient made a rapid recovery and has had no return of symptoms since.

The second patient was admitted to my ward at the Royal Melbourne Hospital six weeks ago, suffering from paresis of the right arm and leg. She gave a history of gradual onset, the first symptom being a loss of skilled movements of the right arm and hand, without loss of power, without any sensory symptoms whatever, and without headache. Examination revealed some paresis of the right arm and leg associated with definite but slight hypertonicity. All skilled movements were very defective in the right arm and hand. When she was told to grasp vigorously with both hands, little defect in power on the right side could be detected; but when she was told to relax the grasp, the left side immediately relaxed while the right remained tightly flexed for an appreciable time and then relaxed gradually. The same slow relaxation to order of the right leg muscles as compared with the left was noticed to a less extent. Slight increase of deep reflexes and diminution of superficial reflexes on the right side were the only other signs. A diagnosis of probable tumour of the left intermediate precentral region was made. X ray examination of the skull showed typical Paget's disease. The patient was allowed to leave hospital, taking a diet rich in calcium and "Ostelin" three times a day, some time before meals. She returned to hospital a week ago, giving a history of fits and increased disability of the right limbs. Soon after admission she had a sudden onset of an extrapyramidal type of fit, without any sensory symptoms, in which the head and eyes turned strongly to the right and the right leg and arm were simultaneously working with the arm well flexed at the elbow and abducted at the shoulder, and the lower limb tensely extended and only flexing slightly during the movements. This condition persisted for ten hours. Morphine in a dose of 0·015 gramme (one-quarter of a grain) was given at once; then 0·12 gramme (two grains) of "Luminal" was given intravenously; four hours later 24 cubic centimetres (six drachms) of paraldehyde were given by the bowel. The fits ceased in ten hours and have not recurred. During the attack the right plantar reflex was typically extensor in type. The condition may be due to local pressure from the Paget's disease; but an associated neoplasm or granuloma may be present. In any case operation is essential. An exploration of the intermediate precentral region is to be undertaken next week.

In considering lesions of the prefrontal region it must be remembered that the mechanism of prefrontal domination is bilateral, and eventually one intact side may almost completely compensate for the opposite side. Therefore it is only in bilateral prefrontal lesions that the typical clinical picture with loss of memory, loss of the power of concentration and of consecutive thought is seen, together with loss of emotional and sexual control. Nevertheless in rapidly developing unilateral prefrontal lesions the negative findings may be definite enough, since the patient shows the same symptoms as a patient with bilateral lesions, but to a less degree. In a rapidly developing tumour or granuloma in this region the main lesion is usually surrounded by a variable amount of œdema, which may at times be sufficiently widespread to cause temporary signs suggestive of an intermediate precentral lesion, with slight apraxia and changes in reflexes on the contralateral side. In slowly developing tumours in this region compensation may be complete and negative signs may be so slight that they are overlooked. Needless to say, subcortical lesions in this region, when the cortex is intact, may produce no symptoms except during periods when œdema round the lesion interferes with cortical function. Two cases are briefly mentioned, one a rapidly developing and the other a very slowly developing lesion.

The first patient came from Hobart with a doctor's letter saying that, up till three months before, the patient had been a bright girl with excellent intellectual development. She then rapidly became irresponsible, neglected her duties and was careless in her manners, and during examination played the fool generally. About the same time headaches became progressively severe and for a month before she was sent for my opinion a gross degree of double optic neuritis had been present. On her arrival at Melbourne, after a rough passage across Bass Strait, she was in a state of complete mental confusion, and any form of sensory examination was impossible, the patient responding to questions with inane giggles. However, there was a difference between the reflexes on the two sides, the right superficial abdominal reflexes being absent and the deep reflexes on that side slightly hyperactive as compared with those on the left side. Both plantar reflexes were typically flexor. After an intravenous injection of hypertonic glucose solution on the following day it was possible to make a reasonably satisfactory sensory examination; all projection areas for sensation were functioning satisfactorily and the reflexes had returned to normal. The degree of optic neuritis was intense and headache was only partially relieved by the hypertonic injection of glucose. The left prefrontal area was explored by Dr. Kilvington and an extensive tumour was palpated, surrounded by a very turgid and œdematous cortex. No attempt at removal was made, but within a few days deep X ray therapy was used. After a stormy month this patient rapidly improved, and at the end of six months her friends judged her to be completely normal. It is now three years since operation and she remains perfectly well. I saw her a month ago in Hobart, and she tells me that she has resumed all her old intellectual interests.

The second patient was sent to me eight years ago with a history that for years her memory had been defective and that for some months she had suffered from bouts of headache, during which her memory was bad and she often found herself clumsy with the right hand. When seen by me she was free from headache; but there was slight double optic neuritis. No neurological signs were detected on several examinations. X ray examination showed a tumour in the prefrontal region, with patchy calcification. A meningioma was suspected. Sir Alan Newton operated and the firm tumour was palpable in the substance of the prefrontal area. No attempt to remove the tumour was made. The patient was much better after this operation. She lost her headaches and her sister informed me that her memory and power of concentration were more satisfactory. Six years later the patient saw me again; she was suffering from severe headaches, and fits which were described as generalized. X ray examination showed a more extensive calcified tumour than that disclosed six years previously. Dr. Coates amputated the tumour and with it the whole of the left prefrontal lobe. The patient made a surprisingly rapid recovery and showed no, even temporary, increase of mental symptoms after operation. She remains well after eighteen months, and her sister tells me that she has better judgement, memory and power of concentration than she has had for a great many years. In this case the condition developed so slowly that ample time was allowed for full compensation to be established from the opposite prefrontal area before the removal of the affected lobe was carried out.

Lesions of the Temporal Lobe.

Lesions of the temporal lobe produce a well-defined clinical picture. The cortex in this region is the projection area for hearing; but, as this is entirely a bilateral mechanism, a unilateral lesion will produce no defect in hearing sounds; but if an acute lesion in this area occurs on the left side in a right-handed person, spoken words lose their significance for the patient and they are mere sounds. Such a lesion produces the greatest defect in the language mechanism and speech is merely jargon, and since the symbols used for thought are not available there is great mental confusion. In a young patient such a lesion is followed by a slow and almost complete compensation of the opposite area, and the speech mechanism returns to normal. The positive symptoms produced by lesions in this area are characteristic and consist of extrapyramidal types of fits which, to the observer, are exactly like those produced by irritation of the parietal lobe and the anterior part of the intermediate precentral region. However, such fits are accompanied by more or less well-organized hallucinations of hearing. If the cortex itself is intact and the lesion is subcortical, the auditory hallucinations may be quite complicated and very clear voices may be heard in conversation and associated visual hallucinations may occur. Such deep temporal lesions are close to the radiations from the posterior limb of the internal capsule and may produce some degree of interference with the optic radiation and with the motor leg fibres on their way to pass into the posterior limb of the capsule. In such cases some degree of hemianopia and paresis of the opposite leg is to be expected. Foerster has shown that if the projection area for hearing is electrically stimulated with the patient under the influence of a local anæsthetic only, typical extrapyramidal fits, with rotation of the head and eyes to the opposite side, associated with auditory hallucinations, occur. Such extrapyramidal movements occur constantly in everyday life, when the head and eyes and body turn involuntarily if some unusual sound is heard, and in the production of such movements the pyramidal tract takes no part.

It is not surprising that no negative (paralytic) symptoms or signs follow the destruction of the temporal extrapyramidal motor area, since its functions would seem to be entirely similar to those exerted by each of the other extrapyramidal motor areas in the parietal region and front portion of the intermediate precentral area. One might expect, perhaps, at least a lag in the automatic turning towards a sudden and unexpected sound. With so many spare areas producing exactly similar sets of movements on stimulation, it is not to be expected that negative symptoms shall result from the destruction of one of these areas.

The function of the extrapyramidal outflow must be largely associated with the maintenance of the upright position. It is clear also that where great physical effort is being used this system plays a large reinforcing part, a fact which may be illustrated in the type of almost universal muscular effort associated with rowing, wrestling, tug-of-war *et cetera*.

I can but briefly refer to the signs produced by lesions of the temporo-sphenoidal lobe. Here again, since taste and smell are completely bilaterally represented no loss of or diminution in activity of these senses is to be expected as the result of a unilateral destructive lesion. Such a lesion may, however, involve that part of the optic radiation which winds round the descending horn of the lateral ventricle deep in the temporo-sphenoidal area on its way to the calcarine area. Such involvement would produce an upper quadrantic hemianopia in the opposite field of vision. It is, however, on the positive signs produced by either subcortical or extracortical lesions in this region that we depend for the full clinical picture. These consist of sensory Jacksonian fits, usually initiated by some pleasant or objectionable taste and smell, and not infrequently, if the lesion be subcortical, there are accompanying visual hallucinations produced by irritation of the optic fibres on their way to the infracalcarine area. With these subjective phenomena the patient drifts into a curious dreamy state and will afterwards describe his surroundings as becoming unreal and fantastic or simply clouded. With such hallucinations of taste and smell, movements of mastication are not infrequently observed. The temporo-sphenoidal symptom complex is so well known that I shall not delay by quoting cases of this condition at this late hour; but I would crave your indulgence while I show three slides of the most remarkable case of cerebral tumour that has ever been under my care.

The patient was first seen by me twenty years ago. He was aged twelve years and had been ill for over three years, with headaches of increasing severity, an increasingly gross paresis of the right leg and arm and of the right side of the face, intense attacks of pain down the whole right side, necessitating morphine, frequent visual hallucinations and some degree of jargon aphasia. About eighteen months before I saw him the aphasia had been more pronounced. At that time, I understand, the optic neuritis was gross. A large decompression operation was carried out, and what was looked upon as a large caseating tuberculoma was found in the parietal region. The surgeon was content with decompression only. After operation the headaches were much less severe and the degree of aphasia gradually improved, and the patient, then aged ten years, learned to write and draw with his left hand. Just before I saw him the headaches had returned with renewed intensity, and he was having almost daily major epileptic attacks, some of which were preceded by intense pain all down the right side, visual hallucinations and vertigo. Examination revealed a typical right-sided spastic hemiplegia associated with diminished sensibility of all kinds, but no actual loss of sensation, except a complete right inferior quadrantic hemianopia. The skiagram (see Figure I) disclosed six main cerebral tumours of great density and a number of smaller ones. All tumours were scattered through the left cerebral hemisphere, none being present on the right side. A diagnosis of multiple cholesteatomata was suggested. Dr. Kilvington operated and scooped out five large tumours containing substance putty-like in appearance and consistency. The patient, after a stormy fortnight, steadily improved and showed no increase in his physical signs except some increased impairment in all forms of sensation over the

ILLUSTRATIONS TO THE ARTICLE BY Dr. S. V. SEWELL.

Figure I.

Figure II.

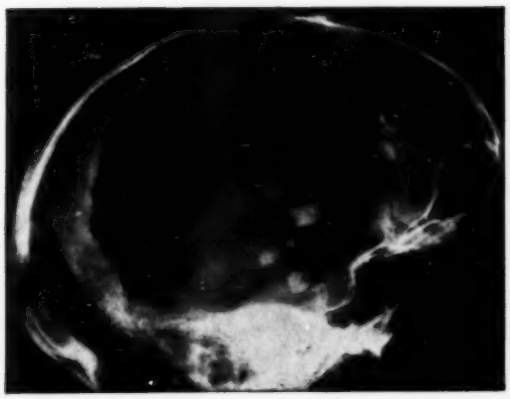

Figure III.

ILLUSTRATIONS TO THE ARTICLE BY DR. W. J. PENFOLD, DR. H. BOYD PENFOLD AND MARY PHILLIPS.

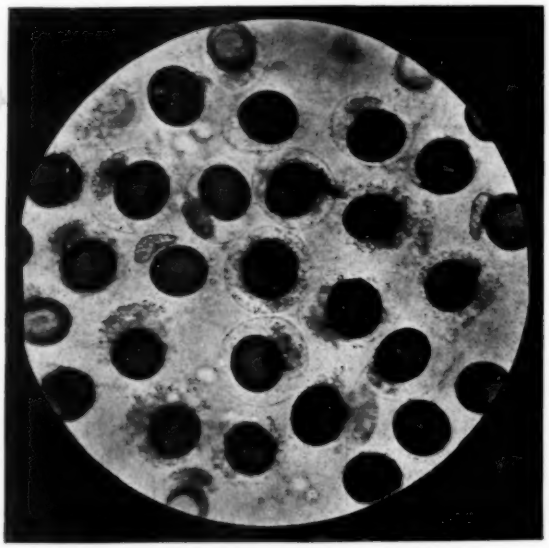

FIGURE I.
Mature *Tænia saginata* ova complete with delicate external shell. (× 240.)

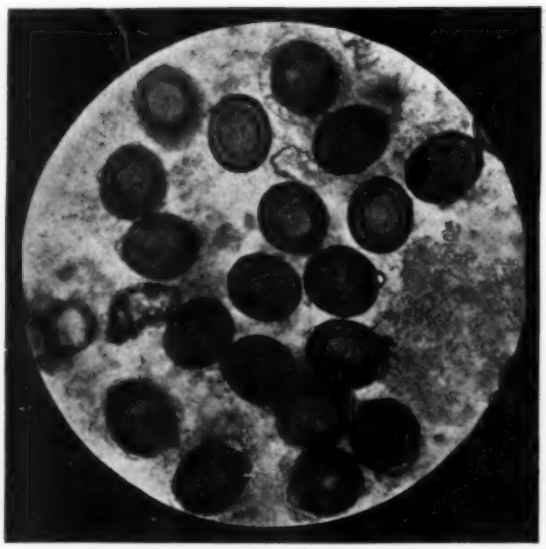

FIGURE II.
Mature *Tænia saginata* ova. Most of the ova are devoid of their external shells. This is their usual appearance in fæces. (× 315.)

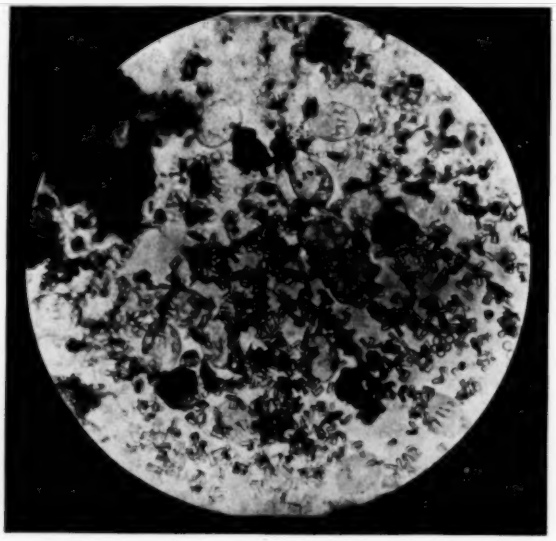

FIGURE III.
Hatched ova. Note (*a*) the free oncospheres, (*b*) the rod-shaped bacillary-like structures which are the remains of the embryophore. (× 315.)

right side. Figure II shows the condition of affairs after the second operation. The slight degree of jargon speech present before operation rapidly cleared up, showing that adequate development of a speech mechanism had occurred on the opposite side. The patient went on with his education and took up commercial art, in which he became most proficient. He continued to improve until four years ago, when he was greatly troubled with attacks of vertigo, with pain all down his right side and occasional unilateral fits, in which the whole right side was simultaneously convulsed without any loss of consciousness. There had been no diminution of intelligence, which was of quite a high order, and at this time he sent me a present of one of his very charming drawings. At operation two large tumours were removed, one superficial and one deep in the parietal lobe. The patient made a rapid recovery and is now a cultured man, thirty-two years old. His speech is quite normal. The finer movements of the right hand have never returned, and all the movements of that limb are typically extrapyramidal in type. The movements of the foot are much more normal, and it is clear that its pyramidal mechanism is only partially affected. This case shows the remarkable degree of compensation that can take place from the other side of the brain despite most extensive brain destruction in a young patient.

I beg to apologize for the absence of any references to the literature, but since this oration was written at sea, entirely without notes or literature, it was impossible to give references.

PEPTIC ULCER: AN ANALYSIS OF TWO HUNDRED AND EIGHTEEN CASES STUDIED AT AUTOPSY.[1]

By R. J. WRIGHT-SMITH.

(From the Walter and Eliza Hall Institute of Research, Melbourne.)

THE problem of peptic ulceration is still keenly exercising the minds of physicians and surgeons, and, despite an enormous amount of work, there still seems to be room for improvement in diagnosis and treatment. We do not appear to have made much progress in elucidating the cause of the disease.

In the present paper an analysis has been made of a large amount of routine autopsy material, with special reference to those ulcers in which hæmorrhage has occurred, and to the difficulties encountered by the surgeon in dealing with this condition. The clinical aspects of the bleeding peptic ulcer are also discussed, with special reference to diagnosis and treatment.

In a series of 4,085 consecutive autopsies performed at the Royal Melbourne Hospital during the years 1927 to 1936, peptic ulceration was found in 218 cases, an incidence which corresponds very closely to that found by Stewart[(1)] in a similar series.

The types of gastric and duodenal ulcers encountered are set out in Table I.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 6, 1937.

TABLE I.
Type Distribution of Peptic Ulcer.

Type of Ulcer.	Number.
Acute gastric	10
Subacute gastric	5
Chronic gastric	97
Acute duodenal	3
Chronic duodenal	95
Chronic gastric and duodenal combined	8
Total	218

Sex Incidence.

Table II shows the sex distribution of the lesions. The ratio of males to females was 3·95 to 1·0. The proportion of males to females in the whole series of autopsies was 1·84 to 1·0. The incidence in males is much greater in all types, and especially in chronic gastric and duodenal ulcer. This agrees with Stewart's findings, except in the case of chronic gastric ulcer, in which he finds that the ratio of males to females is about equal.

TABLE II.
Sex Incidence in Peptic Ulcer.

Type of Ulcer.	Males.	Females.
Acute gastric	6	4
Subacute gastric	3	2
Chronic gastric	81	16
Acute duodenal	3	0
Chronic duodenal	75	20
Combined ulcers	6	2
Total	174	44

Age Incidence.

The age incidence is set out in Table III. The average age for the entire series of ulcers was 57·5 years, that of all types of gastric ulcer being 57·8 years and that of duodenal ulcer being 57·2 years. The youngest patient was aged eighteen years, and presented a chronic gastric ulcer. Acute gastric ulcer did not occur below the age of twenty-five years, and subacute gastric ulcer did not occur below the age of forty-one years. The oldest patient was aged eighty-eight years, and presented a chronic gastric ulcer. Acute duodenal ulcer was not seen in ages below sixty-one years, and was seen once only at the age of eighty years. The youngest patient presenting a chronic duodenal ulcer was aged twenty-five years, and the oldest was aged eighty-four years.

The age incidence of the entire series of autopsies is set out in Table IV. The average age for the entire series was 53·67 years, the youngest patient being aged six months and the oldest one hundred and six years. Rarely are patients under the age of fourteen years admitted to the Royal Melbourne Hospital, and in these autopsies there were only eight patients under the age of eleven years.

The disease was most frequently encountered between the ages of forty and seventy years, reaching a maximum between the ages of fifty and sixty years.

TABLE III.
Age Incidence of Peptic Ulcer.

Age in Years.	10–20.	21–30.	31–40.	41–50.	51–60.	61–70.	71–80.	81–90.	91–100.	Total.
Acute gastric	—	1	1	4	2	2	—	—	—	10
Subacute gastric	—	—	—	—	3	1	1	—	—	5
Chronic gastric	1	2	6	16	34	25	11	2	—	97
Acute duodenal	—	—	—	—	—	1	2	—	—	3
Chronic duodenal	—	8	15	22	27	15	7	1	—	95
Combined ulcer	—	—	—	—	4	1	3	—	—	8
Total	1	11	22	42	70	45	24	3	—	218

TABLE IV.
Age Incidence in 3,522 Consecutive Autopsies.

Age in Years.										
0–10.	11–20.	21–30.	31–40.	41–50.	51–60	61–70.	71–80.	81–90.	91–100.	101–110.
8	149	268	348	586	740	891	457	68	6	1

Site.

In this series the multiple fundus erosions (stigmata of Beneke) have not been considered. The acute gastric ulcers were found chiefly on the "gastric pathway" (Figure I). The chronic gastric ulcers were found chiefly in the same region, but there was a marked increase in the proportion found at the pylorus (Figure II). The chief sites were the pylorus, on or near the lesser curvature and on the lesser curvature, midway between the cardia and the pylorus. This is in accordance with the findings of most authors, and is important from ætiological, pathological and surgical aspects. One here uses the term "lesser curvature" to include the gastric pathway, which really extends for some distance on either side of the lesser curve and widens out as it reaches the pylorus to include the whole circumference of the organ.

FIGURE I.
The site of 10 acute gastric ulcers. C. = cardia; P. = pylorus; A.W. = anterior wall.

The chronic duodenal ulcers were situated in the first part of the duodenum, just beyond the pylorus. They may be adjacent to the pylorus, but they do not extend into the stomach; nor do gastric ulcers extend into the duodenum. Generally, two sites in the first part of the duodenum are favoured: one is on the anterior wall near the antero-superior border (this is the ulcer that perforates); the other is on the posterior wall and is very often adherent to the pancreas (this is the ulcer that gives rise to hæmorrhage). Occasionally one meets with a very large duodenal ulcer which extends from the antero-superior border onto the posterior wall, and which sometimes gives rise to both hæmorrhage and perforation; this occurred once in this series. Acute ulceration occurs generally in the first and second parts of the duodenum.

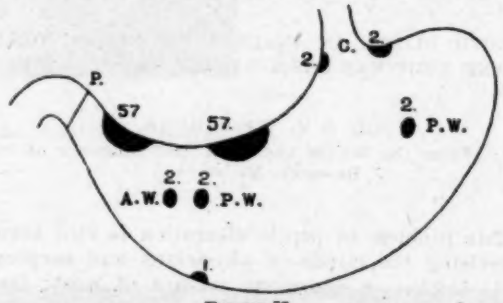

FIGURE II.
The site of 125 chronic gastric ulcers. C. = cardia; P. = pylorus; A.W. = anterior wall; P.W. = posterior wall.

Ulcers Without Reported Symptoms.

There were sixty-three instances of gastric and duodenal ulcers which had given rise to few or no symptoms, death having resulted from some other disease. The great majority of these were unsuspected during life. Thirty-two were gastric ulcers (five acute, five subacute, and twenty-two chronic), and thirty-one were duodenal ulcers (two acute and twenty-nine chronic). The gastric ulcers were multiple in five cases, two being chronic and three acute. The duodenal ulcers were multiple in eight instances, seven being chronic and one acute.

In this group the proportion of males to females in the gastric ulcers was three to one, and the average age was 59·3 years. In the duodenal ulcers the proportion was 2·7 to 1·0, and the average age was 61·4 years.

The causes of death in this group were very varied and had no relation to the ulcers.

Healed Ulcers and Malignant Change.

Healed gastric ulcer was found on eight occasions and healed duodenal ulcer on two occasions. Several ulcers, both gastric and duodenal, showed evidence of healing on microscopic examination, such evidence consisting of epithelization spreading across the base from the edge.

Carcinomatous change was observed on only three occasions, each of the patients being aged fifty to fifty-three years. Two were in males and one was in a female. Two were pyloric ulcers and one was on the lesser curve.

Ulcers Directly Responsible for Death.

Of the ulcers which were directly responsible for the death of the patient, eighty-five were gastric and seventy were duodenal.

Of the gastric ulcers, eighty were of the chronic type and five were of the acute type. Perforation occurred in thirty-three cases (all of chronic ulcers), with general peritonitis in twenty-two cases, hæmorrhage in six cases, subphrenic abscess in five cases, liver abscesses in three cases, pelvic abscess in one case, pyæmia in one case, paralytic ileus in two cases, bronchopneumonia in seven cases and empyema in two cases.

Hæmorrhage without perforation occurred in thirty-five cases, including the five acute ulcers. Bronchopneumonia was present in five cases, lobar pneumonia in two cases, coronary occlusion in two cases and chronic aortic valvulitis in two cases; and pyloric obstruction, hour-glass stomach, cirrhosis of the liver, acute cystitis, acute appendicitis, rheumatic carditis and cholelithiasis were each present in one case.

In seventeen cases of chronic gastric ulcer there was neither perforation nor hæmorrhage. Thirteen of these patients had been operated upon, excision (partial gastrectomy) having been performed in nine instances, infolding in two instances and gastro-enterostomy in two instances. In those operated upon bronchopneumonia was present in eight cases, post-operative hæmorrhage in two cases, leakage of the duodenal stump with general peritonitis in two cases, subphrenic abscess in two cases, liver abscess in one case, lobar pneumonia in one case, gangrene of the lung in one case, and jejunal ulcer with hæmorrhage in one case.

In the four patients not operated upon pyloric obstruction was present in two, bronchopneumonia in three and gastro-colic fistula in one.

In five cases a duodenal ulcer was also present and had perforated on two occasions, while both the gastric and duodenal ulcers perforated on one occasion.

Multiple gastric ulcers were present in fifteen cases. In fourteen cases two ulcers were present, and in one case three ulcers were present.

Of the duodenal ulcers, sixty-nine were of the chronic type and one was of the acute type. Perforation occurred in forty cases, with general peritonitis in thirty-two cases, subphrenic abscess in eleven cases, liver abscess in three cases, paralytic ileus in nine cases, bronchopneumonia in fourteen cases, lobar pneumonia in one case, gangrene of the lung in one case, empyema in six cases, intestinal obstruction in three cases, fæcal fistula in one case and duodenal fistula in one case.

Hæmorrhage without perforation occurred in eighteen cases, with bronchopneumonia in four cases and acute dilatation of the stomach in one case. Other complications included glomerulo-nephritis in three cases, atherosclerosis in three cases, coronary occlusion in one case and hæmophilia in one case.

In two cases both hæmorrhage and perforation had occurred. In one of these two ulcers were present, the anterior one perforating and the posterior one hæmorrhaging. In the other case a large ulcer, previously mentioned, was present, perforation occurring near the antero-superior border and hæmorrhage occurring from the posterior part.

In ten cases there was no perforation or hæmorrhage. Five of these patients were operated upon, gastro-enterostomy being performed in four cases, infolding in one case, entero-enterostomy in two cases, pyloroplasty in one case and appendicectomy in one case.

In those operated upon general peritonitis was present on two occasions, bronchopneumonia on one occasion, paralytic ileus on one occasion and acute duodenitis on one occasion.

Occasionally after partial gastrectomy and gastro-enterostomy the suture line gives way, with leakage of gastric content into the general peritoneal cavity and subsequent peritonitis. The cause of such rupture, even when most careful suture is undertaken, is to be sought in the devitalization of the tissues, especially after hæmorrhage.

Leakage from the duodenal stump may occur if a portion of the pancreas is included in the purse-string suture, with subsequent liberation of trypsin and digestion of the suture.

Excision of the jejunal ulcer was followed by gastric erosions, acute duodenitis and general peritonitis.

In those of this group of patients not operated upon pyloric stenosis was present in one, rupture of the œsophagus below a stricture had occurred into the left pleura in one, and urethral stricture with acute pyelonephritis was present in one.

In this series of seventy duodenal ulcers, gastric ulcer was also present on two occasions and diverticulum of the stomach was present on one occasion. Multiple (two) duodenal ulcers were present in eleven cases and were generally anterior and posterior.

The Bleeding Gastric Ulcer.

Hæmorrhage, severe enough to be the main factor in causing death, occurred in forty-two cases, including six cases of perforation. The average age was fifty-six years. The oldest patient was aged eighty-two years and the youngest was aged eighteen

years. The proportion of males to females was 3·2 to 1·0. The acute ulcers were five in number, there was one subacute ulcer, and chronic ulcers numbered thirty-six. In two cases more than one acute ulcer was present. In nine cases more than one chronic ulcer was present. In five cases of chronic ulcer which had bled, an acute ulcer was also present. In two cases of acute and chronic ulceration a healed gastric ulcer was also present.

In six cases perforation was present. In three of these perforation had occurred in a second ulcer, from which there had been no hæmorrhage.

As would be expected, the majority of bleeding gastric ulcers occurred in the gastric pathway, the largest number being situated on the middle of the lesser curvature, with most of the remainder also on the lesser curvature in the pyloric portion. The two ulcers at the cardia and the four posterior wall ulcers were not readily accessible (Figure III).

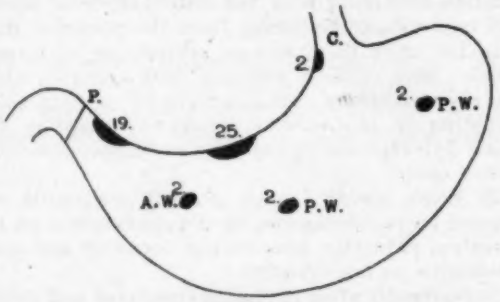

FIGURE III.

The site of the bleeding gastric ulcer in 42 cases. C. = cardia; P. = pylorus; A.W. = anterior wall; P.W. = posterior wall.

The healed ulcers were near the pylorus. Two of the lesser curve (middle) ulcers were large and saddle-shaped. In one case an hour-glass stomach was present.

Nature and Accessibility.

If operation is decided upon, the primary problem of the surgeon is how to determine, with absolute certainty, whether an ulcer is or is not present. If the stomach is not opened, he is forced to rely on a combination of two procedures, inspection of the exterior of the viscus for evidence of peritoneal scarring, and palpation of the viscus in an endeavour actually to feel the ulcer. Both these methods have been tested in this series in the *post mortem* room in an endeavour to assess their reliability. The stomach and duodenum were not opened until a preliminary attempt at localization from the exterior had been made.

As the site of occurrence in over 90% of cases of gastric ulcer is along the lesser curvature, intensive search should first be made of this area.

An ulcer of the stomach will be easily accessible only if it is on the anterior wall or on the lesser curvature near the anterior wall. In the forty-two cases of gastric ulceration with bleeding, peritoneal scarring sufficiently obvious to determine the position of the ulcer was present in only five cases. Sometimes the peritoneal scarring was situated at some distance (one or two inches) from the ulcer. These scars may represent healed ulcers, and Stewart[1] found that scars and ulcers are present with equal frequency. On the other hand, as Aschoff[2] pointed out, scars in the serous coat at some distance from the ulcer may be due to muscular shifting by peristalsis, so one can expect to find them to the oral side of the ulcer. The musculature on this side will taper out and be covered by mucosa, whilst on the pyloric side the muscle is more or less frayed out and terminates in the base.

As most of these ulcers are situated on the lesser curvature, and as the attachment of the gastro-hepatic ligament with its numerous vessels masks the scarring that is nearly always present, visual determination of the site of the ulcer from without is not easily accomplished, and even in *post mortem* material is not reliable. Furthermore, in a fat patient there is always a deposit of fat along the attachment of this ligament to the stomach, which increases the difficulty of seeing any scarring of the peritoneal coat which may be present.

The other methods of fixing the site are palpation and direct vision by opening the stomach.

In palpating the stomach for an ulcer one is again faced with the same difficulty: the great majority of these ulcers are on the lesser curve, a region not easily palpated, owing to the situation of the gastro-hepatic ligament. Possibly a fold of gastric mucosa may be felt and may lead the examiner to believe he is palpating the edge of the ulcer. Against this, of course, is the fact that along the lesser curve the mucosal rugæ are not prominent and are only four in number. Even when handling *post mortem* material it is difficult to palpate the ulcer from without, and the site of the ulcer is frequently missed by this method.

The acute ulcer will show no peritoneal scarring and cannot be palpated from without, as the ulceration is not deep and there is no fibrosis. The funnel-shaped, excavating chronic ulcer will possibly be felt with ease, but large numbers of these ulcers are flat and not deeply excavated, and it is likely that many of these may be missed by palpation. With prolonged medical treatment some healing may have occurred, causing the ulcer to lose its excavating character and making it very difficult to feel as the base approaches the surface and the edges become epithelized.

The third and last method is by direct vision, which, of course, means opening the stomach. The surgeon will make his opening in a situation to give him the best possible view of the lesser curvature, and perhaps after making such an opening he can palpate from within. In this group (the bleeding ulcer) there may be a large or a small amount of blood clot present which will obscure this vision, and he may not feel tempted to remove such clot, though the risk of doing so would not necessarily be great. This method seems to offer the most reliable guide to localizing the ulcer.

Should the ulcer be acute, none of these methods except perhaps the last would be of avail. Further, when more than one ulcer is present, an ulcer which is not bleeding may be isolated and removed, leaving the source of trouble behind.

The Bleeding Duodenal Ulcer.

Hæmorrhage, severe enough to be the main factor in causing death, occurred in twenty cases, including three of perforation. The average age was 47·5 years. The oldest patient was aged sixty-nine years and the youngest patient was aged twenty-two years. The proportion of males to females was 5·6 to 1·0. All ulcers were of the chronic variety. Eighteen ulcers were situated on the posterior wall, one on the antero-superior surface and one on the antero-inferior surface. In six cases two ulcers were present; in one case a chronic gastric ulcer was present as well. In two cases gastro-enterostomy was performed, and in two cases a perforation of an anterior ulcer was repaired. Œdema of the lungs was present in twelve cases, bronchopneumonia in five cases and peritonitis in two cases; and pulmonary abscess, empyema and acute dilatation of the stomach were each present in one case. Associated diseases included atherosclerosis in three cases and chronic nephritis in two cases. Epithelioma of the tongue, hæmophilia, endocarditis, renal calculi, enlarged prostate and hydatid of the liver, the spleen and the omentum were each present in one case.

Nature and Accessibility.

Many of the observations made on the accessibility of the bleeding gastric ulcer apply to this type of duodenal ulcer. For all practical purposes one may assume that the bleeding duodenal ulcer will be situated on the posterior duodenal wall, usually within 2·5 centimetres (one inch) from the pylorus. This ulcer is often situated on a part of the posterior wall which has little or no peritoneal coat, and very often such ulcers adhere to and penetrate the underlying pancreas. Further, the gastro-duodenal artery or its branch, the superior pancreatico-duodenal artery, runs immediately behind that part of the duodenum which is affected by the ulcer.

It will be impossible to see these ulcers from the front or to feel them from the posterior aspect. They may be palpated through the anterior wall, though this method is not without fallacies and perhaps risks; and at least one patient in this series was treated by a purse-string suture through and around normal duodenal mucosa when the ulcer was situated on the lesser curvature of the stomach. Of course, the surgeon may not desire to see the actual ulcer—he may be quite satisfied as to its position and proceed with his operative measures. Should he desire actually to see the ulcer, it is obvious that he will be forced to make an opening in the anterior wall and to view the ulcer from this aspect. As in the gastric ulcer, this would seem to be the most reliable method of localization.

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THE BLEEDING PEPTIC ULCER.[1]

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THE opinions expressed in this paper are based on clinical experience gained in the wards of the Royal Melbourne Hospital under the guidance of Dr. Hume Turnbull, and to a lesser extent on a detailed analysis of fifty consecutive patients who died from bleeding peptic ulcer and were studied at autopsy by Dr. R. J. Wright-Smith. Dr. Wright-Smith[1] has just discussed the pathology of bleeding peptic ulcer, and I propose in this paper to review the subject from a clinical standpoint and to attempt to form some conclusions as to the best methods of treatment. In doing this I have been greatly helped by several excellent contributions to medical literature, notably those by Meulengracht,[2] by Marriott and Kekwick,[3] and by Witts.[4]

Examination of the Patient.

The first task which confronts the physician is to obtain a detailed account of the history. This may be difficult, as the patient usually sinks into a state of mental confusion after severe hæmorrhage from a peptic ulcer. As he improves in mental clarity, further attempts should be made to obtain a more accurate account of any digestive disorders. A study of the records shows that this renewed questioning is often omitted.

The first point of importance is that many patients persistently state that they have never suffered from symptoms typical of peptic ulcer, and the differentiation between gastric and duodenal ulcers is even more difficult. In the present series of fifty cases proven at autopsy, thirteen patients failed to give a history suggestive of peptic ulcer. Every endeavour should be made to obtain the results of any radiological investigations by barium meal which may have been made during the preceding year. In the fifty consecutive patients who died from bleeding peptic ulcer and were studied at autopsy by Dr. Wright-Smith, only six had been examined with a barium meal within six months of the hæmorrhage, and it is of interest to record that not only had all these been reported to have an ulcer by the radiologists, but in every instance the localization of the ulcer was found at autopsy to be correct.

It is not proposed to discuss the differential diagnosis of hæmatemesis, but it should be emphasized that if there are no other obvious reasons for the hæmatemesis, such as congestive

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 6, 1937.

heart failure or hepatic cirrhosis with venous obstruction, a peptic ulcer is the most likely cause, even in the absence of a history of digestive disturbance. It should be recalled that peptic ulcers on the posterior wall not only tend to bleed, but may penetrate into the pancreas, causing severe and persistent abdominal pain. On rare occasions this may lead to an error in diagnosis, cholecystitis or "leaking peptic ulcer" being suspected.

A large hæmorrhage causes pallor, sweating and often fainting. These signs are usually followed by hæmatemesis even if the hæmorrhage is duodenal, though occasionally quite large gastric hæmorrhages may not cause vomiting. Melena is a late manifestation, especially if the patient is constipated.

The physical examination usually yields very little positive information beyond anæmia and possibly dehydration. The site of maximum tenderness may accurately localize the situation of the ulcer, though this sign may also be misleading.

Treatment.

It is proposed, in discussing the problem of treatment, to outline the methods most commonly employed prior to the last three years and then to discuss the rationale of more recent methods. For the sake of convenience, these will be referred to as the "old" and the "new" methods of treatment. It should be emphasized, however, that many physiologists, physicians and surgeons are still opposed to these innovations as being unsound and unphysiological. On the other hand, some of the suggested methods in the "new" treatment have been successfully used by physicians and surgeons for many years.

The "Old" Method of Treatment.

The patient was placed in bed without a pillow, and the foot of the bed was raised about one foot from the ground "to aid the cerebral circulation". A large dose of morphine was given hypodermically and repeated when the patient again became restless. This might require large doses, as he would be thirsty and hungry, since nothing was allowed to be taken by mouth for twenty-four hours or even longer in order to "rest" the stomach. In order to maintain an adequate fluid intake, saline solution or tap water was given *per rectum*, and from time to time "nutrient" enemas were also administered; these usually consisted of glucose-saline solutions. Ice was sucked in order to appease the ever-increasing thirst which was invariably present. After twenty-four hours, or even longer, small amounts of citrated milk were allowed at frequent intervals, and on the third day eggs were included in the diet; thirst and hunger were thus slowly relieved. On the other hand, the patient might lose his appetite and become drowsy, irritable and petulant. Eventually, however, he would probably begin to recover, his mental confusion clearing and the hæmorrhage ceasing. The red cells would be slowly restored by the natural bone marrow response. Usually further hæmorrhages occurred at intervals. The time interval between "bleeds" varied in different patients from a few hours to a week or more. After each hæmorrhage, food by the mouth was withheld for a further twenty-four hours, and if the hæmorrhages were copious and numerous, death was probable. Somerset[6] showed that in 150 consecutive cases of bleeding ulcer admitted to the Royal Melbourne Hospital between 1932 and 1935 the mortality was 23%. The diagnosis was made on a typical history of ulcer, on a positive result to X ray examination, or on the ulcer being found at operation or autopsy.

When the patient became grossly exsanguinated, the advisability of transfusion was discussed, but, owing to the fear of elevating the blood pressure and thus causing a further hæmorrhage by forcing out the clot, this treatment was usually withheld. However, when he later became moribund and death appeared imminent, the blood transfusion was given as a last resort. A pint of citrated blood was injected with some immediate benefit unless a transfusion reaction, consisting of a rigor, collapse and a rise in temperature, occurred either during or shortly after the transfusion. The benefits of the transfusion were, however, short-lived, and death ensued after a few hours, a large terminal hæmatemesis often accompanying the agony of death. The transfusion had proved a failure after all.

In the terminal stages of bleeding peptic ulcer the respirations may be hurried and moist sounds may be heard at the bases. The temperature may rise and bronchopneumonic consolidation may appear, while on rare occasions the patient may succumb to the effects of coronary occlusion. A close study of the fluctuations in the hæmoglobin level or in the red cell count was usually omitted; the appearance of the patient, the occurrence of hæmatemesis, the pulse rate and the blood pressure being relied upon to indicate a further "bleed".

A Criticism of the "Old" Method.—The treatment of recurrent hæmorrhage from peptic ulcer has been studied closely during the past few years, and many important observations have been made which call for radical changes in the principles of treatment. It will first be necessary to review the older methods and to point out the apparently false premises on which these were based.

First, it has been shown by Meulengracht[2] that great harm results from the initial period of starvation. This harm is accentuated by the fact that the patient has often been living on a very restricted diet for several days prior to the hæmorrhage, owing to epigastric pain. In the series of fifty cases, nineteen patients had severe pain for a week or more prior to the first "bleed". It is the ulcer situated on the posterior surface of the duodenum or on the pyloric end of the stomach which tends to bleed copiously, and this is the type which commonly erodes the pancreas, causing considerable pain. The ice which is allowed to be sucked during the period of initial starvation may provide water in small quantities, but it also stimulates gastric secretion, thus aggravating the

ulcerating process. Liberal quantities of water, of food and of salts, especially sodium chloride, are urgently required and, in order to provide these, "nutrient" enemas are given. It has been shown by Corkill[6] and others that although water and sodium chloride are absorbed by the rectum, glucose is not absorbed. It is thus improbable that nourishment can be supplied by rectal injection.

Meulengracht[2] contends that this period of enforced starvation is the principal factor in producing the torpid state into which these patients lapse. I consider that, although it plays an important rôle, the sudden stress of anoxæmia is more dangerous. As the hæmoglobin value falls, the oxygen-carrying capacity of the blood is depleted and every cell in the body suffers from the effects of oxygen starvation. The cells lining the ulcer in particular are thus rendered more vulnerable to the digestive juices.

It is possible that this vulnerability of the anoxæmic cell leads to an extension of the ulcer and to a recurrence of the hæmorrhage. It should be the aim of treatment to maintain the nutrition of the gastro-duodenal mucosa and also to study the happenings further afield, which are of great importance and may even cause death. In the fifty fatal cases of bleeding peptic ulcer studied by Wright-Smith, twenty were complicated by serious disorders elsewhere in the body. Conditions such as chronic nephritis were present prior to the hæmorrhage and were aggravated by the anoxæmia. Other complications, such as bronchopneumonia, primarily produced by anoxæmia and by other profound disorders of body metabolism, were found. As the hæmoglobin value falls, all the cells of the body are subjected to the ill-effects of anoxæmia. This will be particularly severe only if the blood loss is rapid, since the cells appear to possess the faculty of partly adapting themselves to this state of oxygen poverty.

Œdema may occur after a severe hæmatemesis, probably owing to circulatory failure, increased capillary permeability from anoxæmia, and the hypoproteinæmia which follows hæmorrhage; and Wright-Smith[1] has told us that at autopsy the lungs are invariably found to be grossly œdematous, the alveoli being filled with clear watery fluid, which can be squeezed out like water from a sponge. This œdema must contribute to the general anoxæmia of the body by impeding gaseous exchange in the alveoli. It should be recalled that cyanosis cannot appear in the presence of severe anæmia, as there is insufficient hæmoglobin in the blood stream to produce a visible concentration of reduced hæmoglobin. Clinically the respirations may be a little hurried, moist sounds are heard at the lung bases and the air entry is diminished. Pneumonia is often diagnosed wrongly, but true bronchopneumonia may supervene in the water-logged, non-resistant tissues if this debilitated state exists for any length of time, and it is a common cause of death. The distribution of the pulmonary œdema is greatly influenced by gravity, the fluid collecting in the posterior portions of the chest when the patient lies flat in bed.

Finally, it is important to note that in the fifty consecutive autopsies performed by Wright-Smith, widespread atheroma of the arterial system was found in twenty-five. Atheroma tends further to impair the blood supply to the organs by narrowing the arteries. Often the artery which has bled so copiously is thickened and stands out boldly in the floor of the ulcer. Similarly the kidneys and brain suffer, and in the heart there may be some narrowing of the coronary vessels with resulting myocardial failure. Prior to the hæmorrhage these organs may have shown no clinical or biochemical evidence of failure, for under normal conditions they have great reserve power; but atheroma has lessened this reserve and, when the sudden stress of severe anoxæmia is imposed, an alarming degree of failure may occur, thus greatly increasing the gravity of the situation. It is not surprising to find that young people withstand massive hæmorrhages better than the aged.

Anoxæmia is therefore produced by the low hæmoglobin content of the blood, by impaired gaseous exchange in the lungs from pulmonary œdema, and by circulatory failure from cardiac weakness, increased by partial occlusion of the vascular system by atheroma. The erroneous belief that food is absorbed by the rectum leads to starvation; and finally the principal organs concerned with metabolism and excretion are disturbed and there is inadequate removal of the toxic products from the circulation. The rise in blood urea is an index of this. In a previous paper I demonstrated that the changes in the level of the blood urea indicated the changes in reaction of the kidneys to the anoxæmia and often drew attention to a further hæmorrhage. Subsequent studies have confirmed these observations.

If an operation is undertaken at this stage it will be performed on an individual who is a "poor operative risk". It will have to be hurried, and only temporary measures can be adopted for preventing a recurrence of the hæmorrhage. The gastric and duodenal walls will be friable and the suture lines will be slow to heal. In the present series of fifty consecutive autopsies, five patients had been operated upon. Death was due to a recurrence of the hæmorrhage in four cases, and in the fifth sudden death occurred from coronary occlusion the day following operation. Moreover, the stress of the operation will enhance the possibility of complications elsewhere, especially bronchopneumonia.

In what way can this method of treatment be modified?

The "New" Method of Treatment.

The following modifications in the treatment are aimed at lowering the morbidity and mortality from bleeding peptic ulcer.

Diet.—Meulengracht[2] has emphasized the importance of preventing the patient from being starved. He maintains that liberal feeding by mouth, with

a bulky, high caloric and non-traumatizing food, not only supplies nourishment to the already inadequately fed patient, but also aids in arresting hæmorrhage. It is a fallacy to believe that an empty stomach is inactive; hunger is a strong stimulus to gastric motility. Moreover, if ice is placed in the mouth, a powerful reflex stimulus to gastric secretion is set up. The ulcer is churned by an angry sea of gastric peristalsis and is lashed by the waves of concentrated peptic juice. Fresh erosion of the devitalized cells lining the ulcer occurs and hæmorrhage starts once more. As the resistance of the cells falls in the remainder of the stomach wall, widespread gastric erosion may appear. Hæmorrhage becomes almost continuous and death soon ensues. Meulengracht[2] feeds his patients on a bulky diet immediately after the hæmorrhage occurs, and keeps the stomach supplied with food by giving frequent feeds, which include meat balls, broiled chops, omelets, fish balls, vegetable *au gratin*, meat *au gratin*, fish *au gratin*, mashed potatoes, vegetable *purées*, vegetable soups and cream of vegetable. Patients are fed three-hourly and are allowed to have as much food as they want at each meal. Alkaline powders are given to control the pain. We have treated a limited number of patients with a modified form of his diet with encouraging results (see Table I). The patients are comfortable, and only small doses of sedative are necessary to allay their restlessness. They will usually drink large volumes of fluid, but until the drowsy state passes off it may be difficult to entice them to take more than small amounts of the more bulky food. Here the services of a good nurse are important. Sometimes the patient will consider that it is unwise to take solid food, and it is best to discuss the whole plan of treatment with him. Meulengracht's treatment is such a radical change that the physician may feel a little apprehensive as to the result. Before passing judgement one must realize that hæmorrhage will recur in a proportion of cases whether food is given or not. Meulengracht[2] contends that recurrences are less frequent with his *régime*, which seems to be better than the old methods on both practical and theoretical grounds.

After a severe hæmorrhage there is an urgent call on the iron stores of the body to replace the shed

TABLE I.

Modified Ulcer Diet for Patients Suffering from Severe Gastro-Duodenal Hæmorrhage. (After *Meulengracht.*)

Time.	First Day after Hæmatemesis. Approximate Calories, 2,000.	Second Day After Hæmatemesis. Approximate Calories, 2,500.
8 a.m.	5 ounces of milk. Strained oatmeal with milk and cream.	Strained oatmeal with sugar and cream. 2 pieces of *Zwieback* with butter or Stale bread and butter. 5 ounces of milk.
10 a.m.	5 ounces of egg flip. 1 ounce of barley sugar.	5 ounces of egg flip. 1 ounce of barley sugar.
12 noon	1 ounce of strained orange juice. Vegetable *purée* with milk (no meat stock). Simple pudding with cream.	1 ounce of strained orange juice. Vegetable *purée* with milk and "Marmite". Pudding with cream.
2 p.m.	5 ounces of egg flip. 1 ounce of barley sugar.	5 ounces egg flip. 1 ounce of barley sugar. 2 pieces of *Zwieback* and butter or Stale bread and butter.
4 p.m.	1 ounce of strained orange juice. Fruit *purée*. Pudding with cream. 5 ounces of milk.	1 ounce of strained orange juice. Fruit *purée*. Pudding with cream. 5 ounces of milk. 2 pieces of *Zwieback* and butter or Stale bread and butter.
6 p.m.	5 ounces of egg flip. 1 ounce of barley sugar.	5 ounces of egg flip. 1 ounce of barley sugar. 2 pieces of *Zwieback* and butter or Stale bread and butter.
8 p.m.	1 ounce of strained orange juice. Fruit *purée*. Pudding with cream. 5 ounces of milk.	1 ounce of strained orange juice. Fruit *purée*. Pudding with cream. 5 ounces of milk.
Night feeds	(1) 5 ounces of egg flip. (2) 5 ounces of milk.	(1) 5 ounces of egg flip. (2) 5 ounces of milk.

hæmoglobin. Often there has been a hypochromic iron deficiency anæmia existing before the big "bleed" owing to chronic oozing of blood from the ulcer. The oral administration of iron should be started immediately to hasten blood regeneration. It has been found satisfactory to begin with small doses of iron and ammonium citrate and slowly to increase the dose till, at the end of a week, ninety grains are being given daily in divided doses. This will usually prevent any abdominal discomfort from the iron. If the patient is excessively thirsty and dehydrated, extra fluid in the form of normal saline solution is administered rectally by the continuous drip method. A total fluid intake of three litres (five pints) should be aimed at in the first twenty-four hours. At least 1,200 cubic centimetres (two pints) of urine should be passed daily; a diminished output of urine indicates dehydration.

Position in Bed.—It was stressed in a former paper[7] that, except in the case of collapse with gross failure of the circulation, it is not rational to lower the patient's head by elevating the foot of the bed, since this position embarrasses the venous return and is most uncomfortable. If the bed is allowed to remain flat and the head is elevated by one pillow, the patient will be far more comfortable. As the site of maximum œdema will be determined by gravity, it is wise to vary the patient's position from time to time. For part of the day he can lie upon his side, and if the anæmia is not profound he may be put in a semi-Fowler's position for an hour or so. This aids ventilation by relieving the pressure of the abdominal viscera on the diaphragm. Inhalations of carbon dioxide and oxygen can be given every four hours to encourage deep breathing and to guard against pulmonary collapse.

Transfusion.—In my opinion the anoxæmia from hæmoglobin loss is the most destructive agent in severe hæmorrhage from peptic ulcer. It must be accepted that Meulengracht's[2] plan of liberal feeding from the onset has been a great advance and his excellent results are impressive. I am confident that the mortality in the severe cases (with hæmoglobin values under 40%), such as are frequently encountered at the Royal Melbourne Hospital, will still be alarmingly high if Meulengracht's diet is introduced and transfusion is not employed till the patient is moribund. Meulengracht[2] considers that transfusion is required only when the patients have lost large quantities of blood, and of 251 cases of hæmatemesis from peptic ulcer with the hæmoglobin value under 50% he found it necessary in twelve.

Success or failure of treatment cannot be judged wholly by whether the patient lives or dies. Patients may survive after a severe "bleed" and yet be left crippled. The mental acuity may never be regained, and it is reasonable to conclude that some irreparable damage may also have occurred in the heart, kidneys or liver.

So long as the blood loss is not excessive the patient will not die, but the anoxæmia will slowly become less. After a massive single hæmorrhage there are three periods of improvement. First, there is the immediate recovery from shock, when the vasomotor system regains its tone, the blood volume is replaced by tissue fluids and the blood pressure rises. This is incomplete and is followed by the second stage of adaptation of the body cells to the state of anoxæmia. Thirdly, the anoxæmia is slowly relieved by the restoration of the red blood cells as a result of bone marrow activity. On an average the hæmoglobin content will rise from 5% to 10% per week so long as there is no deficiency of iron.

I consider that the greatest advance in the treatment of severe hæmatemesis has been the introduction of massive blood transfusion by Marriott and Kekwick.[3] The benefits obtained by this procedure have been discussed in a former paper.[7] Briefly it may be stated that by the slow administration of blood over a prolonged period large volumes may be given without fear of starting a fresh hæmorrhage; on the contrary, slow transfusion, with consequent elevation of the hæmoglobin value, is the best method of preventing a recurrence of the hæmorrhage from peptic ulcer. There is a popular idea that transfusion will cause a precipitous rise in blood pressure, that the friendly clot will be rudely blown out of the blood vessel in the floor of the ulcer, and that blood will gush forth again into the stomach. Unless excessive, neither blood loss nor blood replacement causes great change in blood pressure. It has already been stated that it is probably a fresh erosion of the devitalized tissues around the ulcer which causes a recurrence of the hæmorrhage, rather than the forcing out of the blood clot. Transfusion, with restoration of the circulation and relief of the anoxæmia, must be the most rational form of treatment, and my experience leads me to advocate it with confidence.

The longer the cells are left in a state of extreme anoxæmia, the longer they will take to recover, and some may be irreparably damaged. Transfusion may fail because it is given too late. In the past these bad results have often been attributed to the baneful effects of the transfusion. In the practice of medicine, as in all other walks of life, one is always loath to accept delay in action as the cause of a bad result. Marriott and Kekwick[3] have vividly described the dramatic improvement which a patient undergoes after a massive blood transfusion. In less than twenty-four hours his colour changes from deathly pallor to a healthy pink, his mental confusion passes away and he feels strong and well. The recovery may not be immediate, as the cells have to recover fully from the anoxæmia, and the rate of recovery is dependent on the extent and duration of this anoxæmia.

Let us visualize the gastro-duodenal mucosa as rejoicing in this state of oxygen abundance. The more severe and prolonged the extreme anæmia and the worse the state of the blood vessels, the slower will be the recovery. The cells have to heal. The recovery of the brain cells is an instructive measure

of this, and useful information will be obtained by simply talking to the patient and testing his memory for past events.

The more rapid the hæmorrhage, the lower the hæmoglobin value, the more extensive the vascular damage from atheroma and the more disordered the metabolism from damaged organs elsewhere in the body, the greater will be the need for relief of the anoxæmia by transfusion. It is therefore impossible to give any concise indications for transfusion. The general plan adopted is to give a transfusion only if the hæmoglobin value has fallen below 50%. When transfusion is performed the hæmoglobin value is elevated to 60% or 70%. The more profound the effects of anoxæmia, the more advisable it is to elevate the hæmoglobin value to high levels, as then a more adequate recovery of the cells may be expected. Chronic nephritis is no contra-indication to transfusion in these cases; the already damaged kidney is in urgent need of an adequate blood supply. Similarly, if an operation is proposed, it is advisable to raise the hæmoglobin value to 70% or higher, when post-operative shock will be lessened and the healing power of the tissues at the site of operation will be greatly enhanced.

Another indication for transfusion is the occurrence of a series of small hæmorrhages from a peptic ulcer, each of which causes a slight depression of the hæmoglobin value. In such a case it is found that a single small transfusion of from 600 to 900 cubic centimetres (one to one and a half pints) of blood will often prevent further hæmorrhage.

The question may be asked why, if it is unnecessary to give a transfusion to a patient with pernicious anæmia in whose blood the hæmoglobin value is reduced to 35%, should so much alarm be occasioned by the same degree of anæmia from a bleeding peptic ulcer? The answer is that in pernicious anæmia adequate liver treatment will restore the hæmoglobin value to 100% and the patient will suffer from no after-effects, first because there is a gradual development of anæmia permitting partial adaptation to anoxæmia, and secondly because there is no risk of a severe hæmorrhage, such as may occur in a bleeding ulcer. There is thus less urgency to obtain a margin of safety.

The technique of transfusion and the precautions taken to avoid reactions have been considered elsewhere,[8][9] so only a brief description will be given here. The method which I am using at present consists in collecting blood from a series of compatible donors into flasks of one and a half litres capacity after the method described by Milroy and Matheson.[10] A large-bore needle[8] is thrust into the vein and the blood flows into the flask, aided if necessary by gentle suction with a reversed Higginson's syringe. Sixty cubic centimetres or two ounces of a 3·8% sodium citrate solution are used as an anticoagulant for 600 cubic centimetres (one pint) of blood. Six hundred to nine hundred cubic centimetres (one to one and a half pints) may be collected from each donor. In the selection of suitable blood donors it should be remembered that all the donors must be of the same group as the recipient.[8][11] Blood from a universal donor, Group IV, Moss (Group *O*)[1] cannot be given in quantities exceeding 600 cubic centimetres (one pint), as the serum will begin to agglutinate the corpuscles of the recipient. The serum of Group IV blood will agglutinate the corpuscles of Groups I, II, III (Groups *AB*, *A*, *B*). Group IV (Group *O*) blood can be given safely in small quantities, such as 600 cubic centimetres (one pint) to recipients of Group I, II or III (Group *AB*, *A* or *B*) because the transfused serum is diluted so rapidly and liberally in the circulation of the recipient that it will fail to produce agglutination. When, however, large volumes of Group IV (Group *O*) blood are given, the percentage of transfused serum will rise and agglutination will begin with grave effects to the patient.

By using Milroy and Matheson's flasks[10] and by changing the delivery tube to a fresh flask when one is empty, satisfactory results have been obtained. By this method the blood of the various donors is not mixed in a common reservoir prior to administration, which is considered to be an advantage, as any tendency to cross-agglutination between them will cause no ill-effects. The flasks are shaken every fifteen minutes to prevent sedimentation of the corpuscles. When the flasks are being changed the rubber delivery tube connected to the thistle funnel is disconnected and attached to the new flask. Before this is done the level of fluid in the dropper is allowed to rise by loosening the cork, in order to enable the air to escape. Then, when the change to the new flask is made, the level of the blood in the dropper again falls, blood thus being sucked over from the new reservoir. In judging the volume of blood to be transfused it is essential to be guided by the level of the hæmoglobin. In a man of 63 kilograms (ten stone) it is found that 600 cubic centimetres (one pint) raise the hæmoglobin value by approximately 10%. Thus, if the hæmoglobin value is 30%, it will take about 2,400 cubic centimetres (four pints) to elevate the hæmoglobin value to 70%, and three or four compatible donors will be required. The blood is administered at the rate of approximately 600 cubic centimetres (one pint) every four hours.

When a patient is first seen it is advisable to make a complete blood examination in order to ascertain the colour index and to exclude the presence of a specific blood disease. Repeated estimations of the hæmoglobin value with an accurate hæmoglobinometer provide the most reliable guide to the severity of the anæmia and to the likelihood of further hæmorrhages. I have found the Sahli model to be simple and accurate,

[1] The classification *AB*, *A*, *B* and *O* included in parentheses is the terminology adopted by the Commission for the Standardization of Sera of the Public Health Committee of the League of Nations.

whereas the Tallquist hæmoglobinometer is not sufficiently accurate to be of help. To treat a case of severe hæmorrhage without accurate hæmoglobin estimations is hazardous, as the patient will often lose a large amount of blood before the physician is confident that a hæmorrhage has occurred. Early diagnosis of hæmorrhage is specially important if early transfusion is to be employed to prevent the hæmoglobin value from sinking below 50%. It must be admitted that the hæmoglobin percentage is an accurate index of blood loss only when there is complete restoration of blood volume by the body fluids. If the hæmorrhage has been excessive or if the patient is dehydrated, relatively high readings are given, but this discrepancy is too small to be of great clinical importance. The pulse rate is a moderately good guide to hæmorrhage, but may be most misleading. I have seen quite large hæmorrhages occur without any alteration in the rate of the pulse. However, apart from the general appearance of the patient, the pulse rate ranks second to hæmoglobin estimations as a useful guide. The blood pressure does not always fall with hæmorrhage, unless this is very rapid or very copious. The patient may be in a state of profound exsanguination before a pronounced fall in the blood pressure takes place, and the manometer has been found to be less informative than the pulse rate.

Indications for Surgery.—The next problem is the desirability of operation to ligate the bleeding vessels. For many years this problem has been hotly debated by both physicians and surgeons. Often the physicians have been more radical in their views than the surgeons. Recently this discussion has been reopened and the problem has been rendered more difficult by the work of Finsterer, Meulengracht, and Marriott and Kekwick. Finsterer has shown that by expert radical surgery a large number of these patients can be cured. But as Witts[4] correctly observes, Finsterer is a genius of gastric surgery, as is shown by the fact that his mortality is 5%.[12] Greater surgical mortality, together with less adequate removal of the ulcerating area, is to be expected in other hands. Conversely, Meulengracht[2] has obtained most excellent results with his high caloric diet and no surgery. Even transfusion is seldom performed in his cases, unless there is extreme exsanguination. He treated 251 cases of hæmorrhage from supposed ulcers of the stomach or duodenum with three deaths. Though Marriott and Kekwick have provided a means of restoring the hæmoglobin to normal levels within twenty-four hours, thus giving excellent preparation for operation, an argument against surgery is the fact that it is difficult to be certain that a peptic ulcer is present when the diagnosis has to be made on the history and physical examination of the patient; and Syme[13] and Wright-Smith[1] have demonstrated the difficulty of finding the ulcer at operation.

I have tried numerous schemes of treatment, varying from operation on every patient as soon as he has received an adequate transfusion to medical treatment alone in spite of repeated hæmorrhages. A compromise represents my present attitude.

The decision as to whether surgery is indicated is based mainly on the number of recurrences of the hæmorrhage. On the patient's admission to hospital the modified Meulengracht diet is given (see Table I) and the hæmoglobin value is estimated every six hours and later every twelve hours until at least four days after the hæmorrhage has apparently stopped. It is then estimated daily for the next week and thereafter less frequently. If the hæmoglobin value is under 50%, the patient receives a transfusion and the hæmoglobin value is elevated to 60% or more. It has been found advisable to represent the various "bleeds" prior to admission to hospital collectively as "Bleed I". If another hæmorrhage occurs in hospital the hæmoglobin value is again elevated to 60% by transfusion and medical treatment is continued. This is "Bleed II". If "Bleed III" should occur and be a small one, medical treatment may still be persisted with; but if it is severe, operation is carried out. If medical treatment is decided upon at this stage, the next "bleed", "Bleed IV", is a clear indication for surgery.

Various other factors influence the decision. A hæmorrhage which rapidly exsanguinates, calls for earlier operation, as a big artery is bleeding and further hæmorrhage may not be readily controlled by transfusion. Also positive X ray evidence of an ulcer, obtained within six months of the hæmorrhage, encourages an earlier operation. However, this ulcer may have healed and a second ulcer, situated elsewhere, may be the cause of the hæmorrhage. Although a definite history of ulcer is encouraging when surgery is contemplated, it should be realized that at least a quarter of the cases of proven ulcer do not have a history of ulcer dyspepsia. Likewise, it is often impossible to differentiate between an acute and a chronic ulcer by the history. Acute ulcers usually cannot be seen or palpated at operation.

Operation therefore may have to be advised when the only clue to the diagnosis of peptic ulcer is hæmorrhage without any other definite cause, such as gross cirrhosis of the liver or heart failure. From Dr. John Horan we shall learn that the recent advances in gastroscopy are encouraging, and this procedure may be of help in diagnosing atypical cases.[14]

In a previous paper it was shown that the height of the blood urea is an aid in deciding the best time to operate, and a series of readings should be taken to study the changes in level. If the blood urea rises greatly after each hæmorrhage and shows only a slight fall after treatment with blood transfusion, food and free fluids, it shows that the renal reserve is being taxed by the anoxæmia. Operation should not be delayed too long, and is best performed from twelve to twenty-four hours after the

transfusion has been given. With regard to the operation itself, as a physician I can only say that by means of transfusion the patient can be rendered fit to withstand any major operation, such as a partial gastrectomy. Most surgeons now agree that the ideal operation for the arrest of hæmorrhage and the cure of the ulcer should be carried out, and that no attention need be paid to the fact that the patient has recently had a severe hæmorrhage. If the patient has not been adequately prepared for such an undertaking, operation should not be performed, for rapid patching operations have usually been failures.

After-Treatment.

Once the hæmorrhage has ceased and the hæmoglobin is restored to normal levels by transfusion and by iron therapy, the treatment resolves itself into the management of a simple peptic ulcer, which is outside the scope of the present paper. It should be emphasized, however, that if the patient has not been operated upon, X ray examination by means of a barium meal should be carried out, and a test meal should be given two or three weeks after occult blood has disappeared from the fæces. It is very unwise to postpone the X ray examination any longer, as a further hæmorrhage may occur, and it is of the greatest importance to know the nature and situation of the ulcer in case surgical intervention is indicated. The diet should contain an abundance of vitamins, and once the immediate stress of the hæmorrhage has passed the patient should have the benefit of sunshine, a wireless set and an occasional cheerful visitor. After the patient has left hospital watch should be kept for a further "ooze" of blood from the ulcer site, by studying the hæmoglobin percentage and by testing the stools for occult blood. It is wise to give an iron tonic for at least two months after discharge from hospital, and appropriate diet, alkalis and regulation of the mode of living are ordered.

Let us set aside the erroneous belief that transfusion causes a recurrence of bleeding; let us be dominated by a desire to supply the cells of the stomach and the cells of the whole body with an abundance of food and oxygen. Though the journey to the goal of successful treatment of bleeding peptic ulcer is fraught with many difficulties and disappointments, let us continue on our way in a spirit of optimism and adventure.

Summary.

1. The problems associated with the bleeding peptic ulcer are discussed. The opinions are based on clinical experience and to a less extent on a detailed analysis of fifty consecutive patients who died from bleeding peptic ulcer and were studied at autopsy by Dr. R. J. Wright-Smith.

2. The difficulty of diagnosing peptic ulcer as the cause of gastro-duodenal hæmorrhage is emphasized. Many patients have never suffered from indigestion.

3. The treatment of bleeding peptic ulcer is discussed and reference is made to the more recent modifications. It is suggested that the relief of anoxæmia by massive blood transfusion as advocated by Marriott and Kekwick is the greatest advance in the treatment. The technique of massive blood transfusion is briefly referred to.

4. Meulengracht's plan, of providing the patient with a liberal quantity of food and so avoiding the initial period of starvation, has proved successful.

5. It is emphasized that repeated hæmoglobin estimations with an accurate hæmoglobinometer affords the most reliable information as to the extent of the blood loss and the presence of subsequent hæmorrhages. Observations of the pulse rate and of the blood pressure are often misleading.

6. The place of surgery in the treatment of bleeding peptic ulcer is referred to, and special reference is made to the difficulty of locating the ulcer at operation. With adequate blood transfusion and a high caloric diet patients may be satisfactorily prepared for a major operation.

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ARTIFICIAL HATCHING OF TÆNIA SAGINATA OVA.

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As a criterion of life of *Tænia saginata* ova, we wished to demonstrate movements of artificially hatched oncospheres. Yoshino[1] (1923) hatched and carefully studied the eggs of *Tænia solium*; Taylor[2] (1926) watched another cestode egg hatch; Ross[3] (1929) succeeded in artificially hatching ova of *Tænia pisiformis* and *Tænia echinococcus*, but, as far as we are aware, no one has attempted to hatch the ova of *Tænia saginata*.

For the hatching of the eggs we usually used artificial gastric juice consisting of an aqueous solution of 1% pepsin and 0·4% hydrochloric acid (pH 1·9), followed by artificial duodenal secretion, which was an aqueous solution of 0·6% pancreatin and 1·25% sodium bicarbonate (pH 8·2). The hatching was done at 39° C. and the eggs were examined in hanging drop preparations under a microscope with a warm stage. The eggs were obtained fresh from ripe segments of *Tænia saginata*. Eggs from the same batch were proved to be viable by animal experiment. The eggs were first spun down in some of the pepsin solution, and after decantation of the supernatant fluid fresh pepsin solution was added, in order that dilution should not occur. The same procedure was followed if the eggs were transferred from pepsin to pancreatin; in each case the solution which was to be added was warmed to 39° C. beforehand.

As a general rule the artificial gastric juice alone was unable to hatch out the oncospheres, but after eighteen hours' exposure it made the eggs look slightly darker. On two occasions, however, after eggs had been four hours in pepsin, a number of free oncospheres were seen. Artificial duodenal secretion alone usually made the eggs moderately black in appearance in six hours. The oncosphere could hardly be distinguished inside the egg. After eighteen hours eggs were sometimes very dark and had a mulberry appearance, and occasionally a free oncosphere was seen.

In order to get a large percentage of oncospheres set free, it was necessary to treat the eggs with artificial gastric secretion, followed by artificial duodenal secretion. This was extremely successful in hatching almost every egg within four and a half hours, namely, four hours in the pepsin solution, followed by ten to thirty minutes in the pancreatin solution. After four hours in the pepsin the eggs looked quite normal, or they might have become slightly darker. When they were transferred to the pancreatin, a change usually began immediately. The whole egg would become black and the outline slightly irregular. Then one or more large cracks appeared, in which the oncosphere could frequently be seen. Gradually the whole of the embryophore broke up into its component rod-shaped bodies and the six-hooked oncosphere was left lying free. It appeared as though hatching had nothing to do with movements of the oncosphere, but was due to the solutions dissolving the cementing substance between the rod-shaped components of the embryophore. In ten minutes there were large numbers of free oncospheres, and in half an hour almost all were set free. If the ova were in the 1% pepsin and 0·4% hydrochloric acid solution for only two hours before being transferred to the pancreatin solution, practically no eggs would hatch, even after twenty hours' exposure to an 0·6% solution of pancreatin. However, when ova were treated with 2% pepsin and 0·4% hydrochloric acid for two hours and then transferred to 1·2% pancreatin, they began to hatch almost immediately, the majority of the oncospheres being liberated in ten minutes to one hour.

It was a fairly simple matter to set free a very large percentage of embryos, but this could just as readily be done with eggs which were known to be non-viable. We, however, wished to use hatching as a means of testing for life by observing movement in the freed embryos. If the hatched embryos showed movement, there was no doubt that they were alive and probably viable. In some experiments 1% to 3% of the hatched embryos showed movements, and in one 33% were moving, but when the experiments were repeated with the same batch of eggs the results regarding movement were inconsistent. Frequently no oncospheres were seen to move. The movements observed never lasted for more than half an hour. Oncospheres that had stopped moving appeared granular and the hooklets lost their symmetrical arrangement. Various methods were tried to get a larger percentage moving or to keep moving those already motile. For instance, free embryos were transferred from the pancreatin solution to ordinary nutrient broth, broth containing 5% ox bile, or non-immune ox serum, all previously warmed to 39° C.

Eggs were also hatched by putting them successively into fresh fluids from different parts of the alimentary canal of the ox. A mixture of fluid from the third or fourth stomach of the ox with artificial gastric secretion, followed by a mixture of fluid from the small intestine with artificial duodenal secretion, also hatched out a large percentage of oncospheres.

The movements of oncospheres were of two types: (*a*) a fairly rapid amœboid movement of the body of the oncosphere, (*b*) a rotating sweeping movement of the hooklets (spines) around their fixed

lower poles. The exact nature of the movements is in some doubt, but the two lateral pairs of hooks rotated in opposite directions, as if the breast stroke in swimming was being performed. Concerning the action of the central pair, none of us has definite views. One function of the movements of the hooklets appeared to be to penetrate the thin membranous envelope closely surrounding the oncosphere. The moving oncospheres would be seen to shed this thin membrane at the posterior end. The oncospheres propelled themselves very slowly and irregularly in the fluid medium.

The accompanying photographs demonstrate the hatching of *Tænia saginata* ova.

Activity of Enzymes Used.

Pepsin (British Pharmacopœia).—The sample of pepsin from which we made the artificial gastric juice (pH = 1·9) dissolved 1,000 times its weight of coagulated egg white in six hours at 40° C., that is, 0·5 cubic centimetre of a 1% solution of the pepsin would dissolve about five grammes of egg white in six hours at 40° C.

Pancreatin.—The strength of the pancreatin used for making the artificial duodenal secretion was such that 0·5 cubic centimetre of a 0·6% solution (pH 8·2) dissolved ten cubic centimetres of a 0·1% solution of casein in fifteen minutes at 40° C.

Summary of Experiments.

The microscopic preparations usually contained approximately 200 ova. All the eggs treated in the following ways were mature and fresh and therefore probably viable. A sample of eggs from the same batch produced cysticercosis in cattle. The procedures, with their corresponding results, may be set out as follows.

Group A: Pepsin-pancreatin sequence (1% and 0·6% respectively, pH 1·9 and 8·2 respectively).

(1) Pepsin .. .. Four hours.
Pancreatin .. Ten minutes, 50% hatched.
Thirty minutes, all hatched, 1% moving.

(2) Pepsin .. .. Four hours.
Pancreatin .. Ten minutes, almost all hatched.
Forty minutes, 1% moving.

(3) Pepsin .. .. Four hours.
Pancreatin .. Ten minutes, almost all hatched, 33% moving; but only 15 oncospheres were in the preparation.
One hour, all movement stopped.

(4), (5) and (6) .. These experiments were performed exactly the same as above and, although almost all the eggs hatched, none was seen to move.

(7) Procedure as above, but after the oncospheres were hatched they were transferred to warm non-immune ox serum. No movements were detected, although the specimen was watched for several hours.

(8) Oncospheres were hatched and then transferred to bacteriological beef broth (pH 7·4) to which ox bile (5%) and glucose (1%) were added. No movements of the oncospheres were detected, although the preparations were observed continuously for three hours.

Group B: Pepsin-pancreatin sequence (double strength, that is, 2% and 1·2% respectively, pH 1·9 and 8·2 respectively).

(1) Pepsin .. .. Two hours.
Pancreatin .. Ten minutes, 75% hatched, no free oncospheres moving, but an unhatched one was actively motile. Almost all hatched in twenty minutes. No movements during one hour in pancreatin.

(2) Pepsin .. .. Two hours.
Pancreatin .. Half an hour, none hatched, but darkened.
One hour, 75% hatched, none moving.
Ten hours later, all hatched (probably examined too late to expect any movements).

(3) Pepsin .. .. Two hours.
Pancreatin .. Examined 16 hours later—all oncospheres hatched and of normal appearance.
Preparation made for photographic purposes only. Not examined early for movements.

(4) Pepsin .. .. Two hours.
Pancreatin .. Examined 16 hours later—all oncospheres hatched and of normal appearance.
Preparation made for photographic purposes only. Not examined early for movements.

Group C: Short exposure to 1% pepsin (pH 1·9), followed by 0·6% pancreatin (pH 8·2).

(1) Pepsin .. .. Half an hour.
Pancreatin .. Twenty hours, darkening but no hatching.

(2) Pepsin .. .. Two hours.
Pancreatin .. Twenty hours, same as (1).

(3) Pepsin .. .. Two hours.
Pancreatin .. Twenty hours, no hatching, slight darkening of embryophore. Oncospheres retracted.

Group D: Long exposure to 1% pepsin (pH 1·9), followed by 0·6% pancreatin (pH 8·2).

(1) Pepsin .. .. Twenty-three hours.
Pancreatin .. One hour, almost all hatched, but no movement.

(2) Pepsin .. .. Eighteen hours, slight darkening.
Pancreatin .. Five hours, 50% of ova more darkened, occasional free oncosphere, rest unchanged.
Ten hours, almost all hatched, no movements observed.

(3) Pepsin .. .. Nineteen hours, slightly dark embryophore.
Pancreatin .. One hour, dark embryophore.
Three hours, almost all eggs hatched, but no movements observed.

Some were transferred to broth, where 3% free oncospheres moved. Some were transferred to non-immune ox serum, in which approximately 3% moved. These movements were probably stimulated by the more suitable pH.

Group E: Long exposure to 1% pepsin (pH 6·0), followed by 0·6% pancreatin (pH 8·2).

(1) Pepsin .. .. Nineteen hours, no change.
Pancreatin .. One hour, no change.
Ten hours, dark embryophores, but no hatching.

(2) Pepsin .. .. Twenty-four hours, very slight darkening.
Pancreatin .. One hour, no change.

Group F: Exposure to 1% pepsin (pH 1·9) alone.

(1), (2), (3) and (4)—
Pepsin .. .. Twenty-four hours, no change except for slight darkening of the embryophore of the majority of ova. A few were very dark.

(5) Pepsin .. .. Four hours, approximately 5% hatched; no movements. Another 5% of eggs were hatching. The remainder of the eggs only slightly darkened in appearance. Hatched and unhatched oncospheres put into 5% bile broth, no change.

(6) Pepsin .. .. Four hours, 1% ova hatched. Remainder darkened.

Group G: Exposure to pancreatin alone.

(1) and (2)—
Pancreatin 0·6% (pH 8·2) Twenty-four hours, slightly darkened.

(3) Pancreatin 0·6% (pH 8·2) Six hours, darkened outline. Seventeen hours, mulberry appearance; 1% of the ova were split, showing the oncospheres. Occasional free oncosphere.

(4) Pancreatin 0·6% (pH 7·2) Same as (3).

(5) and (6)—
Pancreatin 1·2% (pH 8·2) Twenty-four hours, slightly darkened only.

Group H: Miscellaneous.

(1) Hydrochloric acid 0·4% (pH 1·9) .. .. Twenty-four hours, slight darkening of the embryophore.

(2) Sodium bicarbonate 1·25% (pH 8·2) .. Twenty-four hours, darkening of the embryophore, but not so marked as with pancreatin solution.

(3) Tap-water .. Thirty-six hours, no change in appearance.

(4) Sodium hypochlorite (0·5% available chlorine) .. After five minutes' exposure the solution was freely diluted and microscopic preparations were made. Almost all the eggs were hatched. As would be expected, no oncospheres were seen moving, but they were normal in appearance. The hatching process appeared similar to that when digestive juices were used. (Many other hatching experiments were done with sodium hypochlorite, but the one described above gave the quickest hatching without destroying or disfiguring the oncospheres.)

Digestive juices were obtained fresh from the alimentary canal of the ox, immediately after slaughtering. The specimens were numbered according to their origin as follows:

Specimen No. 1, obtained from first stomach, pH 8·01.
Specimen No. 2, obtained from second stomach, pH 7·87.
Specimen No. 3, obtained from third stomach, pH 4·19.
Specimen No. 4, obtained from fourth stomach, pH 6·77.
Specimen No. 5, obtained from small intestine from fourth stomach to common bile duct, pH 6·45.
Specimen No. 6, obtained from small intestine, first seven feet below common bile duct, pH 8·00.
Specimen No. 7, obtained from middle third of small intestine, pH 7·97.
Specimen No. 8, obtained from lower third of small intestine, pH 7·97.

Except for No. 3, which was pale yellow, all the specimens were dark green fluids fairly thick with mucus. The solid material was removed after centrifuging and the clear fluids used. Viable *Tænia saginata* eggs were treated with these mixtures. The several experiments, with the procedures and results, are as follow:

Experiment (a):

No. 1 for two hours.
No. 2 for one hour.
No. 3 for two hours: Ova slightly darkened.
No. 6 for three-quarters of an hour: Ova slightly darker.
No. 7 for half an hour.
No. 8—
After one hour most of the eggs were unchanged, but a small number was hatched and one free oncosphere was moving feebly.
After two hours still only a small number was hatched, but nearly all the eggs were dark and opaque.
After eighteen hours 75% of eggs were hatched. No more seen moving.

Experiment (b):

No. 1 for two hours.
No. 2 for two hours.
No. 3—
After eighteen hours, no change except darkening and slight irregularity of the embryophore. No free oncospheres seen.
After 40 hours, no further change.
No. 5—
After one hour, no change.
After forty-eight hours, almost all the eggs were hatched (that is, hatched in an acid medium).

Experiment (c):

Pepsin 2% + No. 3 (equal parts): Four hours, slightly darker.
Pancreatin 1·2% + No. 8 (equal parts): Five minutes, almost all eggs hatched, none moving.
Non-immune ox serum: No movements.

Experiment (d):

Pepsin 2% (one part) + No. 4 (two parts): Twenty hours, no change. Even the delicate external shells were still intact.
Pancreatin 1·2% (one part) + No. 8 (two parts): After two hours, almost all eggs hatching. Eggs cracked and oncospheres showing.

Experiment (e):

Pepsin 2% + No. 4 (equal parts): Four hours, no change.
Pancreatin 1·2% + No. 8 (equal parts): Three minutes, almost all hatched, but no movements.
Air bubbled through mixture: No movement.

All the experiments just recorded were performed on viable eggs. Eggs from the same batch were proved to produce *Cysticercus bovis* in oxen.

To prove that hatching can be a purely passive process the following experiments were performed.

Eggs were killed by storage for nine months in physiological saline solution at 2° to 5° C. They were proved to be non-viable by feeding experiments on calves after they had been stored for only sixteen and a half weeks.[a]

(*a*) Some of these eggs were hatched by the method described under Group *A*. Most of the oncospheres were similar in appearance to those hatched from viable eggs, but of course no movements were seen.

(*b*) Some of these nine-months-old eggs were hatched by the sodium hypochlorite method described under Group *H* (4).

Experiments (*a*) and (*b*) were repeated with eggs eighteen months old. Similar results were obtained.

Eggs were boiled in 0·85% saline solution for five minutes. We proved by animal experiment that this rendered them non-viable. The oncospheres were retracted so that their diameter was approximately half that of the internal diameter of the embryophore. These ova were hatched by the methods described under (*a*) and (*b*), but the oncospheres would have been difficult to recognize had we not watched them during the process of hatching.

Conclusions.

The following conclusions all refer to *Tænia saginata* ova only.

1. The liberation of oncospheres from their embryonal shells is apparently best undertaken artificially by the exposure of ova to a pepsin-pancreatin sequence or to a solution containing available chlorine, for example sodium hypochlorite ("chlorize").

2. The method of liberating oncospheres by a solution of sodium hypochlorite is apparently new, and it may be applicable to many parasitic ova and so help in their identification. If the sodium hypochlorite solution is too concentrated, the whole of the ovum, including the oncosphere, entirely disintegrates.

3. The liberation of oncospheres from their embryonal shells can take place without any effort on the part of the oncosphere. Dead eggs can be hatched as readily as live eggs.

4. The emergence of the oncosphere from its delicate enveloping membrane is apparently performed by active movements of the oncosphere and its hooklets.

5. In the ox the final stages of hatching of ova in all probability usually occur in the small intestine.

6. Some live oncospheres artificially hatched exhibit the following movements: (*a*) an amœboid movement of the whole embryo; (*b*) a rotating, sweeping movement of the hooklets. Movements (*a*) and/or (*b*) propel the embryo slowly and irregularly in a liquid medium.

Acknowledgement.

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Reports of Cases.

DEGENERATIVE ARTERIOSCLEROSIS.

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THIS case is published as an example of the interesting manifestations of widespread arterial disease. The patient was a man of seventy-three years of age, who was admitted to the Newcastle Hospital with the following history.

He had been troubled with tingling feelings in the left foot for some weeks prior to his admission. The foot became useless when he walked, and cramp-like pains occurred in the left calf. Two weeks prior to his admission he knocked the skin off the dorsum of the fourth and fifth toes of the left foot, and the wounds refused to heal. One week later he completely lost the ability to move his leg.

Examination disclosed that the patient was a very old man, with considerable mental confusion. Purpuric patches were present on the anterior aspect of the leg and dorsum of the foot. There was dry gangrene of the fourth and fifth toes. The left *dorsalis pedis* artery did not pulsate, and the leg and foot were cold to the touch and anæsthetic to all forms of sensation. There was a firm, painful, tender and non-pulsating swelling, which occupied the whole of the left popliteal fossa. The radial and brachial arteries were very tortuous and thickened in a nodular fashion. The blood pressure was not raised and the heart did not appear to be enlarged. The heart sounds were normal. Examination of the urine revealed a faint cloud of albumin, and at times a trace of reducing substance.

Six days after the patient's admission to hospital the leg was amputated above the knee, and at operation it was remarked that the femoral artery was thrombosed at the site of division.

Five days after operation the patient suddenly collapsed and died.

Autopsy disclosed the cause of death as being a rupture of an aneurysm of the descending aorta into the left pleural cavity. The whole of the aorta was the seat of extensive atheromatous change with calcification of the abdominal aorta. There was a commencing aneurysm of the right common iliac artery, and both iliac arteries were atheromatous. The left popliteal artery was the seat of a fusiform aneurysm filled with laminated blood clot. The anterior and posterior tibial arteries were completely occluded by gross thickening of their walls and thrombosis. The heart was normal in size and shape. The kidneys were normal in size, but were very soft and showed some congestion. Unfortunately permission could not be obtained for examination of the cerebral contents.

Microscopic examination of the tibial arteries showed that there was gross deposition of an amorphous laminated substance in the subendothelial zone. There was also deposition of calcium on the internal aspect of the elastic lamina. The muscle coat was replaced by fibrous tissue. The adventitia appeared normal. The lumen of the vessel was occupied by fibrin clot. The wall of the popliteal aneurysm consisted of a thin muscle coat with much fibrous tissue replacement, together with adventitia. The elastic lamina and intima were wanting, and the lumen was filled with thrombus.

Discussion.

The above picture is that of an advanced case of degenerative arteriosclerosis as seen in senile arteritis and diabetic arteritis.

Arterial diseases may be divided into three main types, namely: (i) the degenerative type, (ii) the proliferative type, and (iii) that causing hypertension.

There are two main types of degenerative arteriosclerosis. The first is the type seen in senile and diabetic arteritis, the atheromatous type, whose characteristic distinguishing feature is the deposition of an amorphous substance in the intimal zone. This is a lipoid, mainly a cholesterol ester, and it forms the yellow atheromatous nodules, which may become ulcerated or calcified. In the more progressive lesions the elastic fibres become destroyed and the media atrophied. The adventitia shows no special change. Aneurysm may follow from the giving way of the weakened media. In the medium-sized arteries the atheromatous plaques may occlude the lumina and cause gangrene of the tissues. This type of arteriosclerosis affects the large and medium-sized vessels. The second type of degenerative arteriosclerosis is known as Mönckeberg's disease. In this, calcification follows upon degeneration and fragmentation of the media of the medium-sized vessels.

The proliferative types of arteriosclerosis are inflammatory in origin, and syphilis of the aorta is an example. Histologically the lesion consists of a proliferation of granulation tissue underneath the intima. This results in the appearance of the so-called pearl button nodules,

which contrast with the yellow patches of atheroma. These nodules are eventually replaced by scar tissue. This change is not confined to the intima, but occurs in the media and adventitia as well. The *vasa vasorum* are also affected and are surrounded by inflammatory cells. Buerger's disease is another example of proliferative vascular disease. This attacks the medium-sized vessels, both arterial and venous. There is an infiltration of the media with inflammatory cells. Thrombosis occurs and this becomes organized and vascularized.

All the above types are unassociated with hypertension *per se*. The type causing hypertension attacks the arterioles and is known as arteriolar sclerosis. In this the media of the arterioles becomes hypertrophied and later degenerates into fibrous tissue.

Acknowledgement.

I am indebted to Dr. A. Roberts, honorary surgeon to this hospital, for permission to report this case.

Reviews.

IRELAND AND IRISHMEN.

IN the introduction to "The Oxford Book of Modern Verse, 1892-1935" W. B. Yeats, the Irish poet, tells how Oliver Gogarty, twelve years ago, was captured by his enemies and imprisoned in a deserted house on the edge of the Liffey with every prospect of death, how he managed to get into the garden, and, plunging into the ice-cold December stream under a hail of revolver bullets, "promised it, should it land him in safety, two swans". Yeats was present when this vow was fulfilled. Yeats, whose opinion is surely worth while, looks on Gogarty as one of the great lyric poets of our age. But Gogarty is also a prose writer of the first order. In his "As I Was Walking Down Sackville Street" he gives us what he calls a phantasy in fact,[1] a rare and entrancing book reflecting his cultured and well-stored mind.

Oliver Gogarty is a medical practitioner; he is also a senator in the parliament of the Irish Free State. The title of the book is taken from an old forgotten ballad; and the volume centres round Dublin and its vicissitudes, its happy days, its dark days, and its noble and ignoble men. There is no plot, no set story; rather does the author converse with us on many different subjects. When he tells of the wanton and stupid destruction of noble dwellings—his own and those of his friends—he is not bitter, as he might well be. Nor does he mention his own imprisonment and escape that are described by Yeats. He would have his fellow countrymen subjected to a little discipline: "Discipline is our greatest need. Otherwise we are only yeast to raise other people, among whom we are transplanted, to prominence, if not success."

Gogarty has something to say about the medical student: "Rose and Carless on Surgery, or Osler on Medicine! One or other of these books are all that he can envisage as being representative of the great tradition of medicine, wide as the ages! What are our educators doing? What an account shall they have to render, deprecating imagination and degrading medicine to a rule of thumb! Separating man from the Universe, from his starry bourne. I say to medical students, 'Before perusing Osler, who is quite a modern author and divorced from European tradition, might I not . . . suggest that you dip a little into Paracelsus. . . . Surely your mind is not impervious to the charm of those inadequately appreciated Middle Ages?'" Who dares to gainsay him? On patent medicines he is scathing: "Every Englishman's house is his hospital, particularly the bathroom. Patent medicine is the English patent . . . And most of these patent medicines are taken in the bathroom. The most amazing results are advertised. . . . And the Englishman believes all this . . . His empty churches would be filled twice over by the faith he wastes on the permutations and importance of his lower bowel . . . It saddens me to think of the pent-up faith misdirected that liberated could rise to Heaven in minsters with flying buttress, curious pinnacle and soaring belfry."

But medicine has only a small place in this delightful book. Its chief charm lies in the people whom we meet in its pages: Arthur Griffith, Yeats, Tim Healy (the first Governor-General of the Irish Free State), Talbot Clifton, "Endymion", "Æ", Michael Collins, Augustus John, Senator Phelan, George Moore, Horace Plunkett and many others.

What Gogarty writes of the madness about him in his troubled city might well be the aim of many in this topsy-turvy world: "What a wonderful thing it would be if we could choose and control our madness! Not that awful insanity which King Lear prayed Heaven to withhold: that uprooting of the Reason from storms within as destructive as the ruin of the elements outside: breaking the age-stiffened oak; a brain set adrift by the ingratitude of its own flesh and blood. Not that, sweet Heaven! But to be able to adjust the Reason to the phantasmagoria of Life. And, while maintaining the steadfast lapis lazuli within, to have it played upon by every delicate shading of the weather, deeping its azure now and lighting it again in tone with the luminous and magical haze which is tremulous with life. To be magical as Life itself, and as irresponsible: to be lunatic enough to take the hard edge from knowledge, to be irrational enough to temper Justice with Mercy, and to be able to adjust oneself to the changes in intensity which the waxings and wanings of Reality assume in the shimmerings of its cloud—this would be an ideal adjustment and a poetic opposition to outrageous Fortune."

This most unusual book will be popular among discriminating readers.

[1] "As I Was Going Down Sackville Street: A Fantasy in Fact", by O. St. J. Gogarty. London: Rich and Cowan Limited; Adelaide and Sydney: F. W. Preece and Sons Limited. Medium 8vo, pp. 330. Price: 18s. 6d. net.

THE RORSCHACH METHOD.

THE "Introduction to the Rorschach Method",[1] by Dr. Samuel J. Beck, is monograph number 1 of the American Orthopsychiatric Association. It is largely based on research at the Boston Psychopathic Hospital from 1929 to 1936.

The author describes the rationale of the Rorschach test as follows:

> The underlying function of the Rorschach test is to describe the personality in terms of answers to the questions: (1) "Where does he look for his meanings?" (2) "What meanings does he find?" The attempt is to get a fair sample of how one looks at his environment, and what he sees there. Any procedure which in some sort does this is an approach to personality description, and many techniques make use of the principle. The word association test does so, in that the responses represent meanings, albeit very partial and ephemeral ones, which the stimulus word has for the observer. When the observer is shown pictures and asked to tell what they represent, to make up a "story" about them, it is supposed that what he sees in the pictures, the stories he makes up, reflect interest-trends prominent in his personality. The Rorschach test differs from such procedures in that the symbol (stimulus) has minimal conventionalized meaning, as is not the case with the association-word or the picture. The meanings which the observer finds in the inkblot are more completely functions of his own personality.

This volume provides a manual of test procedures designed to be helpful in its practical application, and consists largely of response records with their interpretations.

[1] "Research Monographs of the American Orthopsychiatric Association. No. I: Introduction to the Rorschach Method. A Manual of Personality Study", by S. J. Beck, Ph.D., with a preface by F. L. Wells, Ph.D.; 1937. Chicago: The American Orthopsychiatric Association. Double crown 8vo, pp. 290.

Part I deals with experiments in healthy adults, the feeble-minded, depressed states, hypomanic conditions, schizophrenics, neurotics, conduct disorders in adults, problem children and mental hygiene cases.

Part II is devoted to details of technique, norms and their interpretation.

The work is extremely technical and quite meaningless for the dilettante reader. It is apparent that much useful information is gleaned through the careful use of the inkblot method, but it is questionable whether such tests will be of much use to the practising psychiatrist. Dr. Beck himself has no illusions on this score. He realizes that its application is art in which "inkblots are instruments in the same sense as the sculptor's chisel or the 'art artist's' pen". The test is not to be compared with a Wassermann test or similar clinical method of approach. Rather is it a complicated means of approach to a *Gestalt* of interrelated categories or traits, each of which must have clear representation in the pattern of response. This may be best realized by reference to an example. Experiment 39 deals with a problem child. Her inkblots are visualized as a butterfly with wings and claws, men with cups, a bat with two feet, a caterpillar, a weasel or something, spiders, ants *et cetera*. The interpretation shows a hemming in the of psychic structure, lack of effective energy, organization ability at the level of mental deficiency, traits of anxiety and apathy *et cetera*.

Congratulations must be extended to Dr. Beck for his clear presentation of a difficult subject, and to the American Orthopsychiatric Association for such an excellently printed work. The type is clear and the arrangement of test scores excellent. It is to be hoped that other monographs will follow.

DERMATOLOGY.

THAT a fourth edition of "Common Skin Diseases", by Dr. A. C. Roxburgh, should be required in three and a half years from the time of publication of the first edition is a true index of its worth as the best shorter text-book on dermatology in the English language.[1]

As a commencement an "Index of Preliminary Diagnosis for Students" is included. This index, if followed according to the instructions, will be found to be an invaluable guide to the diagnosis of the vast majority of dermatoses met with in general practice.

In dealing with cavernous nævi the author lays no stress on the great urgency for the institution of radiation therapy at the earliest possible moment. This is to be regretted in a text-book designed for those practitioners who see these lesions so early, and many of whom are under the false impression that delay in treatment is of no moment.

The increasingly prevalent "occupational dermatitis" is the subject of a short but succinct description. A very useful table of the various types of eruption which may occur following the administration of several drugs, forms part of the chapter on drug eruptions. The two types of dermatitis which may follow the injection of the various arsenobenzols are most clearly differentiated. In view of the increasing use of gold preparations in the treatment of rheumatoid arthritis, the notes on gold dermatitis are of more than dermatological interest.

In our review of the third edition the omission of the occlusive dressing method of treating impetigo was noted. This omission has not been corrected in the section dealing with the treatment of impetigo which is otherwise good.

The author, in common with many other dermatologists, has not found staphylococcal toxoid useful in the treatment of sycosis or recurrent boils.

The popular bogy that X ray therapy in acne leads to scarring is laid to rest.

The half-tone illustrations of the previous editions have been retained, with the exception of four, which have been replaced by better examples, and twenty-six new illustrations have been added. The coloured illustrations of previous editions remain as before and are still not meritorious.

[1] "General Practice Series. Common Skin Diseases", by A. C. Roxburgh, M.A., M.D., B.Ch., F.R.C.P.; Fourth Edition; 1937. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 432, with 3 plates in colour and 165 illustrations in the text. Price: 15s. net.

Notes on Books, Current Journals and New Appliances.

PRESCRIPTIONS.

DR. ESPINE WARD has issued the fourth edition of his book "Favourite Prescriptions".[1] In the preface the author states that he has drawn freely from such well-known text-books as those by Whitla, Hutchison and Collier, Monro and Murrell. The book is interleaved with blank pages, presumably so that the reader can add his own favourite prescriptions to those of the author. While this is not the type of book to which we can give whole-hearted approval, we can imagine that it would be useful to certain practitioners who find it difficult to write complicated prescriptions and who do not wish to rely, as so many do, on the proprietary lines of the drug retailer. Two useful sections of the book are those dealing with the treatment of poisoning and diet tables in certain stated diseases.

MEDICAL SCIENCE FOR THE UNINITIATED.

AN interesting little book, entitled "An Introduction to Medical Science", has been published by the well-known pathologist William Boyd, of the University of Manitoba, Canada.[2] His book, which in our opinion supplies a well-recognized need, is intended for hospital nurses beginning their training, for the pre-medical student "who has not yet commenced his studies at the medical school and who may be wondering what it is all about", and for hospital technicians. The book is divided into three sections; the first deals with some general principles, the second with organs and their diseases, and the third with some practical applications. The subjects are clearly treated and the language is made as simple as possible. For the purposes for which this book has been written we think it is excellent.

FALLACIES EXPLODED.

THE average person has many false notions about health and hygiene and various aspects of human life. The many ridiculous statements that are current about food would alone justify the publication of a book showing them to be false. Dr. August A. Thomen, of the College of Medicine of the New York University, has written an interesting and in places amusing book, in which he explains and corrects many errors and misconceptions pertaining to health and hygiene, major ailments and cancer, minor ailments and remedies, food and diet, mind and the senses, the body, and marriage and death.[3] He also explodes health fallacies derived from superstition and folk-lore. We can well believe that medical practitioners who have read and digested this book will not be at a loss when their patients confront them with old wives' tales and current absurdities.

[1] "Favourite Prescriptions, including Dosage Tables, etc., Hints for Treatment of Poisoning and Diet Tables", by E. Ward, M.D.; Fourth Edition; 1937. London: J. and A. Churchill Limited. Foolscap 8vo. pp. 156. Price: 7s. 6d. net.

[2] "An Introduction to Medical Science", by William Boyd, M.D., M.R.C.P., F.R.C.P., F.R.S.; 1937. Philadelphia: Lea and Febiger. Medium 8vo, pp. 307, with 108 engravings. Price: $3.50 net.

[3] "Don't Believe it! Says the Doctor", by A. A. Thomen, M.D.; 1936. Australia: Angus and Robertson Limited. Demy 8vo, pp. 356. Price: 9s. net.

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References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE CONTROL OF VENEREAL DISEASE.

IN December, 1936, a conference to consider the control of venereal disease was held at Washington, United States of America, under the chairmanship of Dr. Thomas Parran, Surgeon-General of the United States Public Health Service. The proceedings have been published as a supplement to "Venereal Disease Information" of July, 1937, issued by the United States Public Health Service. This report makes interesting reading, especially when considered along with the section dealing with venereal disease in the eighteenth annual report of the Ministry of Health of Great Britain for the year 1936-1937 presented to Parliament in July, 1937.

Mr. Franklin D. Roosevelt, President of the United States of America, in a message to the Washington conference expressed his gratification at the recent increase in public interest in venereal disease; and the Assistant Secretary of the Treasury described the newly-expressed public interest as widespread, far-reaching and intelligent. The same gratification is not felt in Great Britain, for "there is evidence that the public in many parts of the country are still insufficiently informed on the dangers of venereal diseases and the importance of avoiding infection, and of the necessity for early and skilled treatment if it is thought that infection may have been contracted". Whether the difference in the public enlightenment in these two countries is as great as these two statements would lead the reader to believe cannot be determined. Possibly the American observers are too optimistic and the British somewhat pessimistic. The fact remains that one of the subjects discussed at the American conference was public cooperation in the control of syphilis and that a plea was made for adequate provision from the government "to see this fight through to a finish". In Great Britain, on the other hand, the Public Health (Venereal Diseases) Regulations, 1916, seem to leave little to be desired as far as facilities for diagnosis and treatment are concerned. In the first place there are facilities for free diagnosis and treatment and for the supply of "Salvarsan" and its substitutes to doctors for that purpose free of charge. Secondly, there are arrangements by which any medical practitioner may obtain, at the cost of the local authority, a pathological report on any material from a patient thought to be suffering from venereal disease. In Great Britain, it is stated, many persons do not come under treatment because they do not know the dangers and the effects of these diseases, nor the provision that is made for their treatment. This applies particularly to women with gonorrhœa. Moreover, a substantial proportion of infected persons cease to attend at treatment centres before their treatment is complete. In Great Britain the Minister proposes during 1937 to review the existing arrangements in order that the people may receive continuous education and enlightenment. At the American conference one speaker was emphatic that the master problem was the education of the educators. It was necessary, he said, before a start

was made to eradicate syphilis, to eradicate the widely held contempt for mass intelligence, and also to admit the shortcomings that prevailed in the rank and file and "even among the nabobs" of the professions of public health and healing.

The application to Australian conditions of the statements in these two documents would give scope for endless argument; further reference will be made to them on another occasion. The public health authorities in Australia are doing what lies in their power to treat infected persons, and it must be placed to their credit that there is less reticence than formerly about venereal diseases and their train of human wreckage. The rarity with which a case of florid syphilis is seen in present-day hospital practice may perhaps lead medical students to dread its sequelæ much less than their predecessors did twenty-five years ago. The diagnosis of syphilis in its late stages or as a complicating factor in other diseases may be difficult; and this because it is seldom seen in its early stages. There are many problems in the control of syphilis, but perhaps none is greater from the clinician's point of view than the difficulty of bearing in mind the fact that syphilis may complicate any and every other chronic illness. Few clinicians are called upon and fewer perhaps are prepared to give advice in the prophylaxis of syphilis; if they were to think more of prophylaxis, they would be less likely to overlook the surreptitious appearance of syphilis under the cloak of another disease.

Current Comment.

MYASTHENIA GRAVIS.

THE rare diseases of medicine are really more important than they seem. Not only do they provide material for interesting research, but it frequently happens that their study casts a brighter light upon common physiological problems. *Myasthenia gravis* is a case in point. Mary Walker showed that prostigmin, an analogue of physostigmin, was capable of causing great though temporary improvement in the muscle power of the sufferers from this disease, and inquiry then centred round the physiological explanation of this phenomenon. The effects of physostigmin seem to correspond well with the inhibition of the choline esterase of the body, which has been assumed to be unduly active in myasthenia in hydrolysing acetylcholine at the neuro-muscular junctions, and from this reasoning it would appear to be logical to expect an excessive esterase in the serum of myasthenic patients. Most observers have failed to demonstrate such an excess. However, it would seem worth while to try the effect of introducing into the circulation measured quantities of acetylcholine, or one of the more stable choline esters. "Doryl" (carbaminoyl choline chloride) has been tried by F. R. Fraser, M. McGeorge and G. E. Murphy, and they found that the effect of this substance was comparable with that of prostigmin.[1] The effects came on more slowly and were perhaps less striking, but they persisted much longer, so that even the following day the patients had not relapsed into their previous condition of weakness. "Mecholin" (acetyl β-methyl chlorine chloride) was less effective.

It is suggested from these results that the choline in these preparations was utilized at the neuromuscular junctions by the formation of acetylcholine, but further study of their relative choline content made this unlikely. The effect of intra-arterial injection of prostigmin was next tried, and it was found that the muscles of the injected limb tended to recover more rapidly than the muscles of the body generally, though little difference was observed in the behaviour of the choline preparations given in this way. The authors consider that their work confirms the belief that in *myasthenia gravis* there is a defect in the production of acetylcholine, and that the site of this defect is the neuromuscular junction.

Akin to *myasthenia gravis* in some superficial aspects is a still rarer disease, familial periodic paralysis. A study of this curious disease has been made by R. S. Aitken, E. N. Allott, L. I. M. Casterden and Mary Walker.[1] The patient in question used to find that his periodic paralytic attacks began at night, when he awoke to find himself unable to move; he even experienced difficulty in breathing. Consciousness and sensation were unimpaired and the sphincters were unaffected, and the attacks passed off in eight to forty-eight hours. During an attack the muscles were flaccid and reflex activity was lost. The factors observed by the patient as precipitating an attack were curious, for instance, a heavy meal taken late at night, heavy muscular exertion, or excessive eating of sweets.

Investigation proved that the administration of glucose by mouth could regularly evoke attacks, and full analysis of the blood was carried out both during attacks and interval periods. The only definite abnormality observed was a lowering of the serum potassium, which fell from 17·8 milligrammes per 100 cubic centimetres in an interval to 10 to 12 milligrammes per 100 cubic centimetres during an attack.

[1] *Clinical Science Incorporating Heart*, Volume 3, Number 1.
[1] *Ibidem.*

Accordingly, the effect of potassium chloride was investigated during an attack, and it is interesting to read that the affected limbs began to regain their power in ten minutes and were normal within an hour after the administration of this salt. Raising the blood sugar *per se* was found to be powerless to produce an attack of paralysis. The full details of the experiments need not be given here; but the authors conclude that this curious disease is due to a metabolic abnormality, namely, an undue lability of the serum potassium. In normal persons the passage of sugar out of the blood into the tissues is accompanied by a fall in the serum potassium, but in periodic paralysis this phenomenon appears to be exaggerated. These researches may seem to be "highbrow", but, concerned as they are with the normal mechanisms of the body, it may be that they will lead to further understanding of its workings and even yield practical results that cannot yet be foreseen. It is hoped, therefore, that this work may be of some interest to the great class for whom broadcasters have recently invented the word "middlebrow".

SKIN IRRITATION AND CANCER.

IN Australia considerable interest has been evinced regarding the relation of physical agents such as sunlight to the incidence of skin cancer. The views of Australian dermatologists on the subject have been expressed both in these pages and in general medical literature, and it is now interesting to read the account of an inquiry made into the statistical side of cancer incidence in the American Navy. Sigismund Peller and Charles S. Stephenson, writing from the Department of Biology of the School of Hygiene and Public Health in Johns Hopkins University, whose director is the well-known Raymond Pearl, and the Bureau of Medicine and Surgery of the United States Navy, should speak with some authority.[1] They have set out to determine whether a group of men aged from 16 to 45 or 50 years suffer from skin and lip cancer more than the average, bearing in mind that they are intensively exposed to open air, direct sunlight and salt water. Secondly, they ask the question whether the members of this group show the same degree of probability of dying from cancer of the internal organs as corresponding members of the general population, and lastly whether young men cured of cancer of the skin or lip will later show the same disposition to internal cancer as their corresponding groups. The figures used in this inquiry are for the years 1929 to 1936, being based on 875,000 persons-years of active service, and the actual number of cases of cancer was 469. It is found that both morbidity and mortality from skin and lip cancer are very high, there being 8·9 ± 1·0 new cases annually among 100,000 officers and men, and there were 1·25 ± 0·39 deaths from each of these two types of cancer. It is interesting, but not surprising, to learn that out of nine fatal cases of skin cancer occurring in men between the ages of 20 and 49, five lesions were melanomata. The authors think that, owing to the strict medical supervision, which includes annual preventive examinations of a thorough nature, the results of treatment should be better than in the civil population, and that, therefore, in spite of a large probable error in the figures, the high mortality is significant. Comparison of the figures with the findings in the civil population has been made in this regard, and also in connexion with other branches of the inquiry. This comparison, when made between the mortality from skin and lip cancers and other cancers, reveals an extraordinary result, namely, that the mortality rate from other internal cancers is much lower in the United States Navy than might be expected. Further, there is a close numerical relation between the actual increased cancer morbidity of the skin and lip among the naval officers and men and the decreased mortality from all other cancers. The exact application of statistical mathematics may be left to those who wish to examine the details of this article, but the authors' conclusions may now be stated.

The actual frequency of cancer of the skin and lip in the United States Navy is eight times the normal experience, and it seems fair to agree that this must be due to the peculiar conditions of weather and sunlight exposure. Associated with this high morbidity as regards external surface cancers is a greatly reduced morbidity and mortality for cancers of all other types. In other words, about 40% of the men who might be expected to die of an internal carcinoma suffered instead from a skin or lip cancer which was curable. In spite of the "curability" of surface cancers, it must be observed that the mortality from such tumours in the United States Navy was three times higher than in corresponding groups of the civil population. This higher mortality was due to the melanomata and not to any special danger from epitheliomata. If it is indeed true that only a small proportion of persons who have suffered from a surface cancer and who have been cured of it will subsequently produce a tumour of the internal organs, it is a fact of great importance. Obviously, as the authors remark, the problem is worthy of study on a larger scale, for there may be many factors involved. In other respects we would not say that officers and men in the navy could be compared with other population groups, but we should not expect to find such a difference in the incidence of malignant disease. Perhaps the authors go too far in suggesting that exposure to strong skin irritations which are not carcinogenous to internal organs might save a certain proportion of young men from the risk of dying of cancer. In such an inquiry the difficulty of statistical investigation is very evident, not so much on account of fallacies within the groups observed, but because of the troubles encountered in interpretation; but the subject is worthy of further study.

[1] *The American Journal of the Medical Sciences*, September, 1937.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Trichloracetic Acid in the Treatment of Prolapse of the Iris.

J. W. BETTMAN AND H. BARKAN (*American Journal of Ophthalmology*, February, 1937) advocate the use of trichloracetic acid in the treatment of prolapse of the iris. It produces a dry white coagulum, which prevents the entrance of bacteria, whereas the cautery has the opposite effect. A few crystals of trichloracetic acid are put into a medicine glass and one drop of saline solution is added. A toothpick is dipped into the solution, excess is wiped off on the edge of the glass, and the prolapsed iris or the conjunctiva over it is touched gently all over. Almost immediately the prolapsed part of the iris will be seen to turn a milky-white colour. The eye is then irrigated. The application may be repeated daily for a time, then at longer intervals.

A New Operation for Pterygium.

H. MILLER (*American Journal of Ophthalmology*, February, 1937) states that objections to the popular McReynolds operation are, first, that it is contrary to all principles of surgery to bury skin or conjunctiva beneath other tissue and, secondly, that there is a long period of hyperæmia and discharge. He removes the pterygium from the cornea in the usual manner, but incises the upper as well as the lower border. From a point along the inferior border, where conjunctiva and pterygium remain attached, an incision is made downwards and slightly outwards to the lower fornix. Two sutures are placed in the conjunctiva to close the denuded area formerly occupied by the pterygium, but before the sutures are tied the conjunctiva is cut upwards and downwards as close as possible to the limbus, to relieve tension. The incision downwards is now converted into a triangular gap into which the pterygium is inserted and fixed by sutures at the tip and sides. He states that healing occurs by first intention.

Fish Lens Protein and Cataract.

R. F. SHROPSHIRE (*Archives of Ophthalmology*, March, 1937) reports improvement in the vision of cataractous patients following injections of fish lens protein. Of 123 patients treated, 71 gained in visual acuity. In 11 cases no change occurred. In two cases the lens became quite clear in each eye. In a second paper the author presents some chemical studies of the composition of human and fish lenses. It is well established that the human lens consists of an insoluble portion, an albuminoid and three water-soluble, heat-coagulable proteins, the α, β and γ crystallins. Compared with fish lens, the coagulation temperatures of these three substances are in the reverse order. In a comparison of their contents it was noted that in fish lenticular extracts there was an exceptionally high sulphur percentage. The author believes that in fish lens protein there exists a sulphur-bearing amino-acid which, by its action as an auto-oxidising agent, is of therapeutic value.

Retrobulbar Injections of Atropine in Arteriosclerotic Atrophy.

F. C. CORDES (*American Journal of Ophthalmology*, January, 1937) reports the good results obtained by several writers from retrobulbar injections of atropine in optic atrophy. Pillat employs this treatment in the optic atrophy of tabes, vascular disease and retrobulbar neuritis. The arteriosclerotic type offers the best prognosis. The method is that employed in retrobulbar anæsthesia, 0·5 to 1·0 cubic centimetre of a 1 in 100 solution of atropine being injected twice a week. The author reports improvement in a patient of seventy years.

Sarcoid of the Eyelid.

H. C. ERNSTING (*Archives of Ophthalmology*, March, 1937) reports a case of sarcoid of the eyelid of the Boeck type in a young married woman concurrently with erythematous cutaneous lesions on the anterior surface of the legs. The patient, aged twenty-two years, suffered from a growth on the outer half of the right upper eyelid, measuring 2·5 centimetres in length and 4·0 millimetres in thickness. It was situated at the site of an old scar, was bluish-red in colour, and was not painful. X rays showed no changes in the bones, but a shadow in the hilar region of the left lung. The patient was treated with potassium arsenite and X ray therapy of the chest, with satisfactory results. The condition is characterized by the formation of plaques, both cutaneous and subcutaneous. The ætiology is unknown. Some authors consider it of tuberculous origin, others that a filtrable virus is the cause. The prognosis is good.

Lesions of the Fundus in Polycythæmia.

M. COHEN (*Archives of Ophthalmology*, May, 1937) states that in polycythæmia the vision is seldom disturbed, but examination of the fundus may reveal lesions helpful in the diagnosis. The primary form, the cause of which is unknown, is characterized by an increase in the number of red cells and in the hæmoglobin and volume of the blood. The secondary or compensatory form is usually due to congenital heart disease. Of the seven cases of polycythæmia reported, five were of the primary type and two secondary. In the latter there were no complications in the fundi; the retinal veins were distended, slightly tortuous and bluish; the arteries were moderately dilated and purplish; the conjunctivæ were congested. In the primary type distended retinal veins only were seen in three cases; in one there was venous engorgement in one eye associated with œdema of the disk and retinal hæmorrhage, and in the fifth there was bilateral post-neuritic optic atrophy. The distended veins were bordered by a whitish band, due to transudation of plasma. In 1918 the author described a case of polycythæmia with unilateral thrombosis of the central retinal vein.

Modified Subconjunctival Extraction of Cataract.

H. C. ERNSTING (*Archives of Ophthalmology*, April, 1937) describes a method of extraction of cataract that is partially subconjunctival. A conjunctival flap measuring 6·0 by 4·0 millimetres, is made above and laid over the cornea. The conjunctiva on each side of the flap is undermined to the limbus to "3 o'clock" and "9 o'clock". A keratome incision is made above and is enlarged on each side beneath the conjunctiva with scissors. A peripheral or basal iridectomy is then done and the lens extracted. The anterior chamber may be irrigated if necessary. No sutures are required.

Scleromalacia Perforans.

S. P. OAST (*Archives of Ophthalmology*, April, 1937) reports a case of *scleromalacia perforans* similar to those reported by J. Van der Hoeve in 1934. A man, aged seventy-six years, injured his left eye while chopping wood, sustaining a large vitreous hæmorrhage. Fifteen months later there appeared an inflammatory nodule, about 5·0 millimetres in diameter, over the insertion of the superior rectus muscle. It had a yellow necrotic centre. Caseous material was evacuated on incision. After a few weeks the nodule disappeared, as also did the underlying sclera, leaving the bluish uvea visible. A similar nodule appeared near the first and followed the same course. The lesions appeared counter-clockwise until two-thirds of the circumference were involved. Ultra-violet radiation seemed to do more good than any other therapeutic measure.

OTO-RHINO-LARYNGOLOGY.

Tonsillectomy as a Cure for Peritonsillar Abscess.

FORREST W. MERICA (*Archives of Otolaryngology*, May, 1937) discusses tonsillectomy as a cure for peritonsillar abscess. Twenty-one cases, the patients ranging in age from 13

to 56 years, are summarized. There were no complications in any case, and each patient showed immediate improvement. As a rule, general anæsthesia is preferable. An incision is made just through the mucosa, following the anterior pillar and down the posterior pillar. Then, with slight traction on the upper portion of the tonsil, dissection is started in an endeavour to free a portion of the upper pole before the abscess is ruptured. Then, with a suction tip following the knife, the incision is made into the abscess cavity and the abscess is drained completely with the suction tip. This relieves tension immediately and, its upper pole being practically free, the tonsil is easily lifted from its bed; with a small amount of dissection it is loosened from the anterior and posterior pillars, a snare is slipped over the mass, and the tonsil is removed with little difficulty. Occasionally there is rather free bleeding from the pillars or in parts of the fossa. This is a general oozing, and hæmostasis is difficult to obtain with forceps. By the use of an electrocoagulation machine which has previously been set up, this bleeding is immediately controlled, with a minimum amount of trauma, and when this has been used patients seems to have somewhat less post-operative pain. After this procedure is completed and all bleeding is stopped the other tonsil is removed. This makes it unnecessary for the patient to undergo any further surgical treatment at a later date.

Injuries of the Frontal and Ethmoidal Sinuses.

HUGH CAIRNS (*The Journal of Laryngology and Otology*, September, 1937), in a paper dealing with injuries of the frontal and ethmoidal sinuses, endeavours to show by illustrative cases the various ways in which cerebro-spinal rhinorrhœa and intracranial infection may occur after fractures of the frontal and ethmoidal sinuses. These injuries are produced by violent impacts on the forehead or face. In some cases, and particularly in aeroplane accidents, the upper and lower jaws are also fractured, and there is dissolution of the bony connexions of the facial bones to the base of the skull. It is abundantly clear that in fractures of the frontal sinus and cribriform plate there is serious risk of intracranial infection, not only immediately after the accident, but also at a later period, when any new catarrhal infection of the nasal passages may break through barriers weakened by previous injury. Infection usually takes the form of leptomeningitis; but sometimes it may spread through the substance of the brain, producing abscess of the frontal lobe or purulent ependymitis. No statistics are as yet available as to the degree of risk in these cases; but the evidence collected in this paper suggests that the time has come for more active measures to repair the injured dura by means of transfrontal operation and sutures or fascial grafts. It has been shown that the dural defect is usually to be found in the neighbourhood of the cribriform plate, and that access to this region by operation is not a difficult matter. The indications for urgent surgical intervention are clear in cases of injury to the dura during intranasal operation on the ethmoidal and sphenoidal sinuses, and in cases of delayed cerebro-spinal rhinorrhœa and aerocele following head trauma. In the acute stage of frontal and ethmoidal injuries the case for immediate operation has not yet been clearly established. Greater accuracy of diagnosis of the side and site of injury to the cranial base is necessary before immediate operation, with the attendant risk of aggravating shock already present, can be justified. The cases reported in this paper show that much can be done along these lines by more thorough radiography of the anterior cranial fossa. Congenital deficiencies in the cribriform plate may contribute to the production of cerebro-spinal rhinorrhœa, both spontaneous and traumatic, and these also can be disclosed by X rays. It is clear that in the past injuries of the frontal and ethmoidal sinuses have not received the attention they deserve. There is need for more precise and statistical knowledge of the ultimate fate of the patients treated for these injuries and for close collaboration in this field between rhinologists and cranial surgeons.

X Ray Therapy in Malignant Disease of the Throat.

J. STRUTHERS FULTON (*The Journal of Laryngology and Otology*, July, 1937) outlines the technique adopted in the treatment of malignant disease of the throat by external irradiation. Experience has shown that in general the best results, both with X rays and radium, are obtained when the treatment is protracted and carried out at low intensity, and it is a significant fact that the best X ray statistics come from clinics where the X ray intensity is, for various reasons, so low that it approximates to that used in radium therapy. Patients with malignant disease of the throat are treated as a rule over a period of six weeks. The skin reaction begins at about the end of the third week and is maximal at the end of the fifth week; thereafter it subsides during the continuance of the treatment. The reaction is taken to the stage of moist desquamation, and during the reactionary phase the skin surface is dressed with "Lanoline". From the fifth week onwards the patient has difficulty in swallowing and can as a rule swallow only fluids. This phase does not last for more than two weeks at most, and during this period nourishing drinks are prescribed. Many of the patients are treated as outdoor patients entirely; but more debilitated subjects are treated indoors from the fourth week onwards until the reaction has subsided. Severe dryness of the mouth persists for a period of up to one year, due to the action of the radiation on the salivary glands. Glycerine appears to give most relief from this discomfort. The second sequel that is occasionally met with is the development of submental œdema after the treatment. A puffiness develops under the chin and in the submaxillary regions, but subsides entirely after some months. By the employment of six narrow fields, as originally suggested by Lovitt, it has been found possible to carry the dosage to a known lethal level at a depth without permanently damaging the skin and without producing damage to the lungs. A disappearance of the primary lesion is obtained in practically all cases. The technique is in the process of evolution, but it is certainly producing encouraging results. When one is dealing with a fungating tumour involving the whole of the œsophagus, healing is almost bound to occur by fibrous tissue, and in a number of cases this has actually occurred and has proved a troublesome sequel. It is hoped that this complication may be met in the future by a scheme of intubation during the reactionary phase.

The Treatment of Intrinsic Laryngeal Cancer.

F. HOLT DIGGLE (*The Journal of Laryngology and Otology*, July, 1937) discusses the treatment of intrinsic laryngeal cancer, and concludes that the operation of laryngo-fissure in carefully selected cases has stood the test of time. Although knowledge of the application of radiotherapy has increased considerably within recent years, much remains to be learnt. Statistical tables, however carefully presented, do not always represent the true picture; for this cannot always be expressed by figures and words, but can be learnt only from personal experience. Time alone will show whether the results at present achieved by the Finzi-Harmer irradiation operation are as lasting as those following laryngo-fissure. One has to remember the possibility of radium necrosis, which in a few cases may be long delayed and may even end fatally with no recurrence of malignant disease. Such deaths should always be recorded as "due to malignant disease", even in the absence of recurrence. Treatment by irradiation involves the careful consideration, by a skilled radiotherapist, of many factors, such as the type of growth, its radio-sensitivity, and the dose required if a successful result, with the minimum amount of discomfort to the patient, is to be obtained. If the radiation treatment of intrinsic laryngeal cancer is to progress, it must be given opportunities in early lesions; but its future will depend upon the closest cooperation between the radiotherapist and the trained laryngologist, who alone can decide the site and extent of the lesion and whether it is suitable for treatment.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on October 6, 1937, PROFESSOR R. MARSHALL ALLAN, the President, in the chair.

Peptic Ulcer.

DR. R. J. WRIGHT-SMITH read a paper entitled: "Peptic Ulcer: An Analysis of 218 Cases Studied at Autopsy" (see page 1027).

DR. IAN WOOD read a paper entitled: "The Bleeding Peptic Ulcer" (see page 1031).

DR. HUME TURNBULL opened the discussion from the medical point of view. He said that he had had the good fortune to be able to watch Dr. Wood's work, and that he wished to express his admiration for it. If anyone wanted many long night sessions and comparatively little sleep, he could commend to their notice an investigation on hæmatemesis. Dr. Wood had been good enough to speak of his "guidance", but really he had been the learner, and all the credit for the investigations was Dr. Wood's. Dr. Turnbull said that one of the things which first impressed him was the realization that he did not know the signs of severe hæmorrhage. He feared that he was not alone in expecting that a large hæmorrhage would show itself by an increased pulse rate, and that the pulse would rise if bleeding continued; but in many cases the pulse rate on the patient's admission to hospital had been in the region of 96, and, although further bleeding occurred, had fallen after a few hours, so that it was not unusual to find a pulse rate of 72 with a hæmoglobin value of 45% or even less. That the blood pressure did not fall greatly at first was better known, but even so it was strikingly little affected until very late in the picture, so that when the blood pressure really did fall noticeably, the condition had advanced to a dangerous point. Blalock's work had made this point clear.

Dr. Turnbull said that his next difficulty, and the chief one, was diagnosis. He had gradually abandoned all his cherished beliefs in the symptoms of peptic ulcer, or at least in the value of these classical symptoms in making a diagnosis when the patient had lost a considerable amount of blood. Several patients had denied anything more than a slight epigastric discomfort, and had insisted that this was not related to meals; and they had confirmed this story even when they were restored to a reasonable mental clarity. The most consistent symptom seemed to be relief on taking food, though this might be very short lived. As Dr. Wood had said, the absence of other recognizable cause for bleeding had to be accepted as establishing a probable diagnosis of ulcer. For this reason it was very important that every endeavour should be made to recognize peptic ulcer before bleeding occurred, and, if possible, to localize it; and then to make sure that the patient knew the diagnosis, preferably by having it in writing, so that in the event of subsequent hæmatemesis the task of any other physician might be simplified. However, Dr. Wright-Smith had shown that fatal bleeding might occur even in acute ulcers, and severe bleeding in these was probably not uncommon. Whether surgery would be able to reduce the mortality from large hæmorrhages was still to be proved, though Dr. Turnbull felt that it would, if the surgeon could be told where the ulcer was, for the fact that a chronic ulcer bled while the patient was under medical treatment showed that this was not adequate in some cases.

Dr. Turnbull said that he was quite convinced that if the patient was treated at once and the hæmoglobin level was kept up by massive transfusion, the surgeon could be assured that operation would not of itself increase appreciably the risk to the patient. He had not seen one of Dr. Wood's patients whose prospects of recovery he felt had been diminished by operation, even when the surgeon had not found a condition which he could remedy. Such a boast would not be justified if transfusion was delayed or if frequent bleedings were permitted. Dr. Turnbull was very sanguine as to the results of reasonable feeding of the patient after a hæmorrhage, for he had never been able to believe in the theory of starvation; and the practice was so unpleasant that for many years he had fed his patients almost from the beginning. He had not, however, attempted anything so complete and rational as Meulengracht taught, and he felt confident that this would be a big change for the better. Many patients, especially those with many cares and a limited income, took inadequate amounts of vitamins as part of an ulcer diet, even when they were back at work.

Dr. Wright-Smith's paper showed how many difficulties confronted the poor surgeon, even if an accurate diagnosis had been arrived at.

He did not wish it to be thought that the surgical treatment of peptic ulcer, or even of bleeding peptic ulcer, was being advocated. Unsatisfactory as the medical treatment might be, there seemed little doubt that promiscuous surgical treatment was much worse. It was believed, however, that by the means described, patients could be brought to a state in which operation was practicable and reasonably safe, and that if diagnosis could be improved by another stage, operation would save some persons who would otherwise die. Performance of operation by a skilled surgeon, even if no ulcer was found in a position where it could be effectively dealt with, would not jeopardize the patient's life.

SIR ALAN NEWTON discussed the surgical aspects of the subject. He said that he offered to Dr. Wright-Smith and to Dr. Ian Wood his sincere congratulations upon the work they had done on this very difficult problem, and assured them that their papers would have a great effect in clarifying the views upon the treatment of patients with bleeding peptic ulcer. He hoped that both of them would be encouraged to continue their work, to regard the papers they had just given as progress reports, and to address those present again on this subject at some future time. He trusted that at this future meeting it would be out of place for the Science Committee once more to exhibit a grim sense of humour by giving the place of honour in the discussion to the pathologist; but he trusted also that the surgeon would continue to occupy the last position, both in treatment and in discussion.

Sir Alan Newton went on to say that enthusiasm for surgical treatment of bleeding peptic ulcers had waxed and waned at intervals during the previous two decades. Shortly after the War it was thought that the problem of arrest of hæmorrhage could best be solved by surgical means. When the mortality of these operations was at last assessed, the pendulum swung the other way and surgeons were loath to operate at all in the presence of this complication. More recently the bad results of medical treatment, particularly of what Dr. Wood had described as the "old" medical treatment, had caused some surgeons to reconsider this decision, under the belief that their mortality, bad as it was, could not be worse and might be better than the medical mortality. Sir Alan believed that, if physicians could produce results from the "new" medical treatment even approximating to those reported by Meulengracht, surgeons would be only too glad to withdraw from this field, except when, as Dr. Ian Wood had suggested, the occurrence of a large third hæmorrhage or of a fourth hæmorrhage indicated clearly that medical treatment had failed. Before operating in such cases the surgeon was entitled to ask that the patient should be restored to the condition of a "reasonable risk" by blood transfusion. Sir Alan Newton thought that the surgeon was also justified in inquiring whether there had been any undue delay in replacing the blood lost in any of the former hæmorrhages, because his own experience had led him to believe that, if an elderly patient had had a prolonged period of anoxæmia after hæmorrhage, operation on him was not a reasonable risk, despite the fact that his hæmoglobin had been raised by subsequent transfusion to a

reasonable level. If there had been a prolonged period of anoxæmia, the surgeon was justified in refusing operation. This impasse could be avoided only by early transfusion in cases of severe hæmorrhage.

The surgical treatment of bleeding peptic ulcer ought surely to be identical with that of chronic peptic ulcer: gastrectomy for chronic gastric ulcer and gastro-jejunostomy for contracting duodenal ulcer with obstruction. It was undesirable to operate, if this could be avoided, upon a non-contracting duodenal ulcer; but, if operation was undertaken, gastrectomy was indicated for this condition. If, owing to technical difficulties, gastrectomy was contraindicated, the exclusion operation described by Devine should be performed. The chief difficulty with which the surgeon was confronted in the case of bleeding ulcers was that, as Dr. Wright-Smith had shown, it was usually impossible at operation to see or to palpate the ulcer. In such cases Sir Alan thought that opening the stomach to search for the ulcer was a time-wasting procedure, and that it was preferable, providing that no other cause of the bleeding, such as hepatic cirrhosis, was revealed, to assume that an ulcer was present and to perform gastrectomy. There would, of course, be a material mortality from this procedure, but if surgery was reserved for medical failures, some patients would be saved who otherwise would have died. There was everything to gain and nothing to lose.

Dr. Ian Wood had referred to an instrument, the gastroscope, which might revolutionize gastric surgery. Since an efficient instrument had been designed, it should be possible to ascertain whether an ulcer was present by this means and, further, to discover whether the ulcer was responding to treatment. It might be possible later on to arrest hæmorrhage by coagulation diathermy by modifying this instrument for the purpose. It was impossible to visualize bladder surgery at the present time without the cystoscope, and Sir Alan hoped that the gastroscope would prove equally useful.

DR. JOHN HORAN considered the gastroscopic findings in cases of peptic ulcer. He showed slides to demonstrate the Wolf-Schindler flexible gastroscope, the technique employed in flexible-tube gastroscopy and the various depths of introduction. He stated that since the objective of the instrument had a viewing angle of 80°, a large part of the stomach wall was brought into the visual field. Dr. Horan showed a slide to demonstrate the *musculus sphincter antri*, the angulus, the pyloric antrum and the pylorus, as seen by the gastroscopist.

Dr. Horan said that the prepyloric ulcer, which was situated immediately behind the angulus, was hidden from view. Such an ulcer, however, might produce Henning's indirect sign of tenting of the angulus. The gastroscopic picture of the benign ulcer was striking. The benign gastric ulcer was oval, and there was an abrupt transition between the white or yellow colour of the base of the ulcer and the surrounding orange-red mucosa; occasionally blood could be seen running from an ulcer to the mucous lake at the most dependent part of the greater curvature. Hæmorrhagic spots occurring on the lesser curvature were a frequent accompaniment of gastric ulcer. Schindler had observed patients with such hæmorrhagic spots, who subsequently developed true gastric ulcer, and he considered that there was a definite connexion between them. This opinion, however, was not accepted by Faber and Konjetzny, who maintained that a true gastric ulcer arose from an erosive gastritis.

Dr. Horan showed slides to illustrate chronic hypertrophic gastritis. In this condition the mucous membrane had a velvety appearance, and the high-lights were diminished or absent. The mucosal folds became enlarged and nodular. At times small ulcerations appeared on the crests of the nodes and were frequently the source of hæmorrhage.

DR. G. R. A. SYME, who had operated on twelve of the patients referred to by the previous speakers, commented on the extraordinarily good condition of the patients when they were handed to him; only four of the twelve patients had failed to recover. He had found local anæsthesia of extreme value, and had noted that soon after operation the general condition of the patient was better than would have been expected if the operation had been conducted under general anæsthesia. The cause of death was in one case, that he had been unable to find the ulcer; in another case he had encountered adhesions; extreme technical difficulties had arisen in the other cases too, such as high position of the ulcer and breaking down of the sutures. Dr. Syme mentioned with satisfaction the use of a transverse abdominal incision followed by the division of both *recti abdominis* muscles; by this means a much better exposure was possible without much traction. A small injection (two or three cubic centimetres) of "Evipan" had been given while he sewed up the wound.

Dr. Syme discussed the selection of patients for operation, preluding his remarks by saying that the surgeon was not anxious to operate on these patients. When there was no history of ulcer, but the bleeding was continuing, the surgeon had to make up his mind to perform gastrectomy and to remove the ulcer-bearing portion of the stomach. When there was a definite history of ulcer it was usual to find the ulcer easily and to excise it. In the cases of two of the patients who had died, Dr. Syme had thought afterwards that it might have been better had he performed partial gastrectomy; and in another of the fatal cases the Devine exclusion operation or a posterior gastro-enterostomy might have been more satisfactory than the excision of the ulcer, which was on the posterior wall of the first part of the duodenum and was hard to expose and required too much traction. Finsterer, Dr. Syme stated, had reported a 5% mortality by operating early, by operating on "medical" ulcers and by performing gastrectomy as a routine measure.

DR. C. H. OSBORN, after thanking the readers of the papers, said that the whole problem of the treatment of hæmatemesis was not completely solved, and that the surgeon had to exercise all the ingenuity he could when facing the problem of the bleeding ulcer; the results were bad and the mortality was high. He drew attention to one or two additional measures that could be used before the patients were handed to the surgeon. On the assumption that the ulcer was continuing to bleed because the clot was being digested away and that normal healing was not taking place, and on the further assumption that this digestion was associated with hydrochloric acid and pepsin and that the digestion could be avoided, Meulengracht had advocated the use of bland food to absorb the acid. In addition, by means of the continuous alkali drip, it could be neutralized continuously. Some patients did not withstand ordinary aluminium hydroxide, which efficiently neutralized acid and stopped further digestion. The other measure which was used to put off the necessity for operation concerned the principle of rest for the stomach and the ulcer. In a proved case of gastric ulcer it was a measure of value to pass the duodenal tube into the duodenum and another tube into the stomach. Dr. Osborn said that the Levine tube was well borne through the nose and was better than Ryle's tube. Continuous suction of the stomach contents was carried out in this way. Dr. Osborn also stated that, when dealing surgically with a chronic, large, penetrating duodenal ulcer, it was useless to perform anything but gastrectomy, including the removal of the ulcer; the exclusion operation had a lower mortality rate and saved the added mortality of attempting to remove the first part of the duodenum.

DR. W. W. OSTERMEYER suggested that calcium carbonate might be used, in the hope that it would neutralize the acid and minimize the tendency to bleed. He also mentioned that adrenaline might be given by mouth for its hæmostatic property. He made the comment that the modern methods of giving food to these patients were distinctly reminiscent of the old Lenharts diet.

DR. A. E. COATES said that operating for bleeding ulcers was a specialized branch of surgery, in which Dr. Syme had made himself proficient. Dr. Coates had had relatively little experience, but he recalled one instance in which

the patient had died on the tenth day after operation; the abdominal wound had fallen apart, and he correlated this with the fact that the patient had been in an anoxæmic state for over forty-eight hours before the operation. He expressed the opinion that Ogilvy's amputation of the stomach just on the gastric side was the safest type of amputation for a general surgeon not specially trained in this class of work.

DR. W. J. NEWING asked Dr. Ian Wood whether he had been able to establish any criteria which enabled him to decide to carry out a transfusion of blood. He thought that he could remember that Witts had given figures to the effect that a transfusion should be given if the hæmoglobin percentage fell below 47% and the blood urea rose above 100 grammes per hundred cubic centimetres of blood. Dr. Newing also asked whether the increased use of blood transfusions had resulted in fewer operations. He remembered that in that room about two years earlier Dr. Gordon Taylor, of London, had advocated surgery in the early stage of hæmatemesis.

DR. IVAN MAXWELL said that he wished to refer to some of the biochemical points. Dr. Turnbull had mentioned that the blood pressure did not fall greatly with loss of blood. It might be of interest for him to state that, as the result of animal experimentation in anæmia, this fact had been established. There was a greater output from the suprarenal gland in hæmorrhage, and therefore some vaso-constriction, and in addition the fibrinogen content of the blood increased, which tended to stop the hæmorrhage. Dr. Ostermeyer had referred to treatment with alkalis; certain types of colloids helped to stop the bleeding, but neutralization with sodium bicarbonate induced a further flow of hydrochloric acid. Dr. Meakins had advocated the administration of alkalis for the relief of pain, but they had to rely on adsorption by the colloids or by a high protein diet.

PROFESSOR R. MARSHALL ALLAN congratulated the speakers on the complete way in which the subject had been presented and discussed, and called upon the opening speakers to close the discussion.

Dr. Wood, in reply, said that the discussion had been informative and helpful. With reference to what Dr. Osborn had said about the use of tubes in the stomach and the duodenum, Dr. Wood said that they had been rather averse to the introduction of more than one new thing at a time; they intended in the next stage of the work to try to leave the surgeon out and to see what measure of success could be obtained by relying on careful dieting and transfusions of blood. He hoped that Dr. Osborn would pursue the method of using the tube and give a report on his results later; it was quite likely that this method would be included in the future in the work at the Royal Melbourne Hospital. In reply to Dr. Ostermeyer, Dr. Wood said that it was realized that at the Royal Melbourne Hospital they had had only the bad cases of hæmatemesis to cope with; even then less than half of the patients had required transfusion. There were certainly many patients with hæmatemesis of milder degree who did not gain admission to the wards of the hospital. Dr. Wood informed Dr. Newing that, if the hæmoglobin value was estimated to be over 50%, they did not give a transfusion of blood, and they could usually get the patients through the illness without this form of treatment. If a patient had a series of small hæmorrhages, they preferred to give a transfusion of blood if the hæmoglobin value was down to 40%. Dr. Wood said that he had noted that Witts laid great stress on the blood pressure and advocated transfusion of blood if the systolic blood pressure dropped below 90 millimetres of mercury; but, as Dr. Maxwell had said, the body had remarkable powers of preventing this fall of blood pressure, though much blood was lost. Dr. Wood said that the rise in blood urea depended greatly on the rapidity of the hæmorrhage and the duration of anoxæmia; sometimes a patient would weather the storm after prolonged anoxæmia, but this state had to be regarded as a grave risk. In conclusion, Dr. Wood stated that they should have confidence that transfusion of blood would elevate some of the patients from the dangerous zone, and that it would not promote hæmorrhage but, on the other hand, would help to prevent it.

A MEETING of the Victorian Branch of the British Medical Association was held at Warragul on October 2, 1937, DR. J. P. MAJOR, the Senior Vice-President, in the chair.

Poliomyelitis.

DR. F. V. G. SCHOLES gave an address on the epidemiology and nature of poliomyelitis, with special reference to the characters of the present epidemic in Victoria. He said that poliomyelitis was due to the transmission from person to person of a virus which depended for its existence on human beings. This phenomenon of parasite and host existed throughout Nature, and one could envisage a gradual adaptation towards a perfect balance, a state of amity, ideal for both, where neither harmed the other. In the case of bacteria the phenomenon could be studied quite easily, and Dr. Scholes could quote quite a number of examples. For instance, in anthrax they had a terrific foe which with a little entry into the body would overwhelm and kill without any possibility of defence; in tuberculosis and leprosy, perhaps owing to insolubility, little opposition was evoked and extensive lesions gradually developed, finally either killing mechanically or opening the way to other organisms to do so in their own way. With streptococci, diphtheria bacilli and pneumococci a gradual process showed itself and had developed over hundreds of years towards perfect equilibrium; they were getting partial immunity in Victoria already, and Dr. Scholes thought that these organisms would ultimately live in amity with the host. Finally, he said, there were harmless flora existing in plants and animals in a state of equilibrium.

The viruses presented a different proposition; their requirements were so exacting and their existence so frail that they were difficult to study; unless they were pathogenic, little could be learned of them. Dr. Scholes quoted the extreme case of Asiatic smallpox, in which there was no quarter on either side in this country and in temperate zones; here smallpox killed, and killed quite quickly. The viruses of measles, rubella and varicella occupied an intermediate position, and at the other extreme Dr. Scholes placed the virus of poliomyelitis. Poliomyelitis was a disease to which there was apparently quite a good deal of immunity, and in which there was not a very hard actual warfare. They must presume that there existed innumerable harmless viruses, some of which caused little febriculæ and others no symptoms at all. The frequency of occurrence of subclinical attacks in a population gave some indication of the distance that was reached towards equilibrium. In smallpox and in explosive diseases like measles an enanthem and an exanthem were distinguishable; cells were killed and desquamated and the whole thing was over quickly. Subclinical attacks were rare in measles and smallpox; they were commoner in varicella, rubella and mumps; but they were very common in poliomyelitis. The possible factors in determining the subclinical nature of an attack were: (i) The dosage of the infecting organism, if the defence was strong and there was enough localization. Dr. Scholes said that they were on safe ground in talking in this way of dosage in the bacterial diseases, but the ground was not so safe in poliomyelitis. (ii) Implantation on an unsuitable site, such as implantation of the virus of poliomyelitis on a tonsil; although it was thought that the virus of poliomyelitis could thrive only in nerve cells, Dr. Scholes thought it possible that it might excite a little immunity response if implanted on the tonsil. (iii) Previous acquaintance under such favourable conditions as a small dose implanted on an unsuitable site might, on next acquaintance, result in the production of a subclinical attack, even if implanted on a susceptible surface and in a dose which, without the previous acquaintance, would have resulted in a clinical attack. If the first acquaintance made was made under unfavourable conditions and in big dosage,

the way was open to a fight to the death quickly between the virus and the body.

Dr. Scholes put forward the thesis that the virus could multiply and thrive only in living body cells and in no other conditions. Further, some viruses could multiply and thrive only in a certain type of living body cell. The virus of poliomyelitis, they were beginning to think, could multiply and thrive only in a nerve cell. He did not know that they were quite safe in accepting the last statement fully. They had in other diseases, caused by viruses not strictly termed neurotropic, effects on nerves and nervous tissue such as the encephalitis of measles, varicella and vaccinia. These viruses were not truly regarded as neurotropic, and when one spoke of the virus of poliomyelitis as neurotropic it might not be safe to say that the virus could live and multiply only in nerve cells, though he was confident that this was the case in 99% of instances. Generally speaking, when a virus entered a body cell, one or the other had to die, and that pretty quickly. Dr. Scholes regarded this thesis as important and its truth had to be settled by the experimenters. Generally it was the virus and not the body cells that died; it was most interesting to see in microscopic preparations, such as those of Dr. Reginald Webster, perfectly healthy nerve cells in apposition to dead or dying cells. The biochemists and experimenters must find out for them what exactly took place to enable the virus to get inside a cell envelope and exactly what happened after it was in the cell. Dr. Scholes was satisfied that there could not be any truce; viruses could exist, but could not multiply, in dead cells, but they could not exist for long; they could survive in mucus and in fluid for a short period. More study was needed of the pustules of smallpox and varicella. Viruses could kill cells, but did not often kill the body, and death in virus diseases should be regarded as accidental; if the exanthem in measles was in the chink of the glottis and produced suffocation or if it clogged up the inmost recesses of the lungs, death occurred thus accidentally. Similarly the poliomyelitis virus killed accidentally if it happened to pick out certain vital nerve cells such as some of those about the floor of the fourth ventricle or cells subtending muscles which carried out vital functions. With the exception of these accidental deaths the virus of poliomyelitis did not kill. Dr. Scholes said that deaths from smallpox intrigued him; he understood death from streptococcal pyæmia, but had not been able to work out exactly what killed the patients in the comparatively early state of smallpox; it might be the result of the entrance of a virus; but the other viruses did not kill the body except by accident.

Dr. Scholes proceeded to the discussion of the process of infection by a virus. He thought that infection by viruses was quite a different proposition from infection by bacteria; while, no doubt, the general principles of response and reaction were the same, still there appeared to be considerable differences in detail of methods both of attack and defence. He referred then to the power of causing infection, the opportunity of causing infection and the time of infectivity. He said that the capability of causing infection resided in the fact that there must be dead or dying cells containing living virus that must be shed off from an infected person's body; the opportunity of causing infection was not so big a one as was the case with bacteria which were very infectious; infection generally occurred by droplet infection, and the possibility of droplet infection depended on quite a number of factors. In measles, poliomyelitis, mumps and other virus diseases, the virus was cast off in cells by coughing, sneezing, blowing or spitting. It was obvious that a person to be infected must be within reasonable distance of a person who was infecting him; the amount of virus per cubic inch of air diminished with distance and so the chance of the virus landing became smaller, and the dangerous distance could be worked out. Other conditions affecting the transmission of the virus would include the nature of the atmosphere, whether the exposed person and the infecting person were inside a room or in the open air, and, if inside a room, whether the windows were shut or, in the open air, whether the wind was blowing. In the case of rubella, infection did not occur in an adjoining room or in the opposite corner of a ward; if the windows were open there was hardly a virus known that could be transmitted by air; if the viruses fell to the floor or went up to the roof they became harmless. In the case of measles transmission there was a violent cough and relatively big masses of mucus could be sent further than would be the case, say, in mumps or poliomyelitis. Considerations of the opportunities for droplet transmission already had a place in the practical management of infectious patients, Dr. Scholes explained; in the management of measles in a ward the beds, tables and desks had to be spaced further apart, for instance, than in a ward of patients with mumps.

Dr. Scholes then discussed the time factor, and said that patients with virus diseases were infectious only while the dead cells were cast off and while the virus was still living. In practically all of these conditions the time during which infection was possible was very short; each year it seemed to Dr. Scholes that his conception of the duration seemed shorter, and he hazarded that, for practical purposes, in another five years or so the period would be regarded as very short indeed. He summarized these statements by repeating that there could be no truce between the virus and the cell; one of them must die. For a person to be infected the conditions must be favourable; he must be susceptible; the virus must be cast off from the infector who must be in sufficiently close proximity to infect the other person, and that infector had only a short time in which to do the damage. In addition, it was necessary to consider whether it was possible to have equilibrium; whether there was a possibility of a person being a "carrier" or "harbourer".

Dr. Scholes then discussed what happened when an epidemic of virus diseases occurred, and why after it fizzled out no cases occurred for a while, except odd stray cases, and then none whatever. He said that in any community, whether a house, a home, a village, a town, or a continent, it was important to consider what had become of the virus when, after a time, no further cases occurred. He stated that at the time of the meeting one could affirm that there was no smallpox in Australia; there was no measles in Victoria, and there was no poliomyelitis in the room. In thinking of a community like Melbourne, with a population of about a million people living in an area of about one hundred square miles, it was of importance to consider what had happened to the virus when there was no longer any case of virus disease in Melbourne. The time taken for the disappearance of a virus from such a community varied according to the disease. When after an epidemic of measles in Melbourne an interval of, say, six weeks occurred without a case, they were justified in asking what had happened to the virus. In rubella, with the probability of many mild or subclinical cases, the interval must be lengthened, and with poliomyelitis, in which the proportion of subclinical cases was very large indeed, still further. But even with poliomyelitis, after eight, nine, ten or twelve months during which no more cases occurred, it was appropriate to ask what had happened to the virus. The first possibility was that the virus was fully active and circulating, but not producing any clinical cases. He believed this was the orthodox view; but it was just not possible, and he had discarded it long before the outbreak in 1937. He preferred to use the term "isolated case" rather than "sporadic case" for the occasional clinical appearance of poliomyelitis without obvious source of infection and when no epidemic was known to be occurring. The last case or subclinical case must have had the opportunity to pass infection on to a susceptible person. Even in bubonic plague the infected flea had to find a susceptible human being, bite him, and then vomit into the wound, and the flea had only a few hours to live. A consideration of the subject of the infective period led them to conclude that it was of brief duration and the incubation period could not possibly be short. With each fresh transmission the virus found increasing difficulty in lighting upon a susceptible person and passing on the infection; knowing the incubation period and the size of

the exposed population, it was possible to calculate approximately the time that it took for an epidemic to work itself out.

The second concept was that the virus was active, but not circulating, which was impossible because if it was cast off it was still circulating. One then had to consider whether the virus was circulating but relatively inactive. This was an attractive theory to Dr. Scholes. He elaborated it by suggesting that the virus could persist, producing immunizing doses and only in certain people producing "sporadic cases". Ten to fifteen years earlier he had thought that that was what happened; but the laboratory workers had said that the quality of toxicity was not lightly changed over a short period, though a change might happen over centuries. It was necessary, therefore, to keep an open mind about this theory until the "sporadic cases" had been made the subject of intensive study. During the ten or eleven months before the present epidemic there had been only one case recorded in Victoria; it was down on the books as such, and it might have been an exotic case, say, from Queensland or anywhere else. He had from time to time during many years attempted to trace isolated cases of virus disease; it was the usual thing for the investigation to fizzle out; often the diagnosis was revised or the reported case was not a recent one. The only other two possibilities that could be put forward were that the virus was so very very slightly active that they did not know whether it was alive or not; or that the virus was dead; that there was not any. Dr. Scholes was prepared to accept only this last possibility. After a time, then, there was no virus in the community and the disease could start again only by the importation of fresh living virus. The orthodox view was that an epidemic of poliomyelitis was not the manifestation of the activity of imported virus, but of heightened activity of latent virus. He said that there were some communities, strongly urbanized ones, where poliomyelitis was endemic. It had appeared in Greenland in 1914, and not again until 1932, when it reappeared and hit the people pretty badly; particularly those born since 1914. Did anyone in the room believe that the virus had been lying latent in that Greenland community for all those years, not producing any clinical cases? There had been no measles in Melbourne for well over a year. Did they not believe with him that it could never reappear in Melbourne unless reintroduced? The present epidemic of poliomyelitis had been introduced to Victoria from without; there was strong circumstantial evidence of this with a lot of cumulative corroborative facts. From the first cases occurring in June, 1937, the 428 clinical cases which had been reported since had descended. For the first 100 cases good evidence had been obtained, particularly by the work of Dr. Merrilees, of a transmission from person to person or group to group, with date and place, but after that all the paths interlocked, though, generally speaking, in very many cases the origin and date were known. The readiness with which the transmission had been traced was one of the special features of interest in the epidemic, and was allied to the second feature of unusually high transmissibility. The geographical progression pointed to transmission by children, and indicated that adult transmission was negligible except in a number of individual cases; 600,000 adults had been travelling in all directions across Melbourne, but the disease had travelled steadily from an area infected originally by leakage into neighbouring suburbs, radiating from classes of school children and from kindergartens and along tram routes used by school children. Many thousands of adults had been contaminated, many less of them had been infected, but, with individual exceptions, they were not infectious. It was a reasonable hypothesis, in his opinion, that the life of the virus on the mucous membranes of a high-grade immune person like himself was only momentary, if it could occur at all; even if he was infectious, he had to have the opportunity to infect non-immune persons in such a very short time. The millions and millions of contacts among adults which had occurred, if even a small proportion had resulted in transmission of the disease, would have caused the whole area to have been infected at least two months before the meeting. He concluded, therefore, that, compared with the immense number of possible infectors, infection by adults was a very small factor indeed. Another feature of interest in the present epidemic was that the disease had been affecting the higher levels of the central nervous system; poliomyelitis was becoming a more serious disease in its effects, producing a larger number of lesions of serious import. They took it that the mode of spread was from the olfactory hairs, to which the virus attached itself, along the olfactory bulb into the thalamus and hypothalamic areas into the mid-brain and medulla, and more or less simultaneously to the posterior root ganglia and the posterior horn cells. It had been definitely established that the route was along and inside non-myelinated axones, and when the virus arrived at a block it was relayed into new nerve cells. It was rather difficult to see why the virus in so many cases went down to the lower limb cells and left untouched so many important cells. In one epidemic six hundred lower limb cases occurred and only forty upper limb and a few basal cases. In the present epidemic they were seeing paralyses of almost any level almost in equal quantities; facial, palatal, pharyngeal, respiratory, and skeletal, neck, back and abdomen and both limbs had all been involved fairly equally, and the higher levels had been hit badly.

Dr. Scholes thought that the symptoms were explainable entirely on the basis of the action of the virus purely on nervous tissues. He had found it difficult to explain the real "sickness" of the patients on the theory of axonal spread, but the pathologists said it could be explained in that manner. There was early involvement behind the olfactory bulb and before the virus reached the mid-brain, and there was involvement of the sympathetic nervous system. The localized hyperæsthesia was due to the fact that the posterior root ganglia and the posterior horn cells were involved quite early. There was no longer any need to invoke the theory of systemic spread to explain the symptoms, nor was there any reason to hope to cure people with convalescent serum or anything like that.

In conclusion, Dr. Scholes classified the cases as: (i) those with affection of respiratory, pharyngeal or cardiac centres; (ii) severe cases with extensive paralysis in which the bad signs at the onset were fear, fever, vomiting, headache and the early development of tremor; (iii) extensive moderate or light cases in which all or some limbs were involved early, but recovery occurred to a greater or less extent; (iv) a small group in which the paralyses were mild and localized; (v) subclinical cases of vague illness and known contact; and (vi) abortive cases which might start severely and stop at any stage, even to the moment of reaching the anterior horn cells or even after initial tremor or other severe symptoms. Time did not permit of the discussion of signs, cell counts, differential diagnosis, use of the respirators and many other features of interest, and Dr. Scholes preferred to leave the questions of splinting and after-care to the orthopædists.

DR. F. KINGSLEY NORRIS thanked Dr. Scholes for his address, and said that it had been a rare opportunity for them to hear such an authoritative crystallized talk on epidemiology. In the country, Dr. Norris said, poliomyelitis presented a very different problem to that in the town; it consisted of a salting of little immunizing attacks. He wished to refer particularly to some of the difficulties created in practice by the epidemic. There were many odd conditions being encountered in private practice. It had been regarded as essential that poliomyelitis should be diagnosed immediately while in the preparalytic stage; but the early diagnosis really did not matter to the patient, though it was important to allow of segregation of contacts to curtail the spread of the disease. Though the diagnosis was fairly easy in most cases, in some it was very difficult. There was an extraordinary amount of pharyngitis about; some of it might be due to infection with the virus of poliomyelitis. There were some strange types of myalgia associated with fever and some *erythema nodosum*. Above all there was the newspaper disease, which was prone to affect particularly girls between nine and fifteen years of age. These patients had all the symptoms in the clinical

picture of poliomyelitis, with the exception of elevation of temperature and paralysis. He had adopted the dictum that where there was no fever there was no poliomyelitis. There had been lots of opportunities of determining the incubation period, Dr. Norris said, and he instanced an example in which almost to the hour the incubation period had been ten days. The patients were not being treated with serum, and without prejudicing the patient it was quite reasonable to continue observation for some days before making the diagnosis definitely, though in any suspicious circumstances it was desirable that the patient should be isolated and the contacts segregated without delay. Dr. Norris pointed out that it was not possible to influence susceptibility, but that by avoiding gatherings of children as much as possible dosage of virus transmitted was minimized, and by avoiding fatigue, if the child was infected, paralysis was likely to be less extensive. They had noted the association of untoward fatigue just preceding the onset of symptoms and severe paralysis or even death of the patient. Dr. Norris also commented on the situation in Kew, where there had not been a reported case for a month. The health officer had been vigilant and had used strong and powerful propaganda against unnecessary gathering together of children, combined with the advocacy of the use of mechanical aids to minimize dosage of droplet infection.

DR. H. C. MCLORINAN said that it had been their experience at the Infectious Diseases Hospital at Fairfield that the clinical stages corresponded fairly well with the theory of axonal spread. The first symptoms on or about the tenth day after exposure in numbers of instances had been vomiting ascribable to hypothalamic infection, and dilatation of the pupils which occurred was evidence of true sympathetic stimulation. The first stage was a psychic or apprehensive stage which was followed by a sensory stage due to stimulation and pain of posterior nerve root origin. The paralytic stage followed on or about the fourteenth day as a rule, and was succeeded by the stage of recovery. With reference to the results of examinations of the cerebro-spinal fluid, Dr. McLorinan said that in most cases the cells were increased in number, but that in certain cases the cell response had been very slow and had not been appreciable till after paralysis had occurred. It was not the result of meningeal irritation, but could be looked upon as a spilling over of cells from the perivascular spaces, the fluid of which communicated by a devious route with the cerebro-spinal fluid, thus accounting for the cell response as a late phenomenon. He added that the differential diagnosis, which had been very worrying to the parents and to general practitioners, had been a matter of great interest to him and to the members of the resident medical staff at the hospital. Coughs and sore throats were not common, but they had had patients with retropharyngeal abscess and cervical adenitis; it was useful to remember that the head was held erect in poliomyelitis and forward in cervical adenitis. Another patient had manifested the spine sign and neck stiffness which had turned out to be due to a small abscess below the scapula.

DR. STANLEY WILLIAMS recommended those that had not done so to read the paper by Dr. Mostyn Powell published recently in THE MEDICAL JOURNAL OF AUSTRALIA, in which could be found a very good account of the symptomatology of poliomyelitis in the present epidemic. The apprehensiveness and the absence of irritability, except in children around the age of three years, and the fine tremor in the hand or leg, localized rather than general, were noteworthy observations. The persistence of neck stiffness rather than the presence of it was a point to note in the differential diagnosis from acute pneumonia or *otitis media*. Since the publication of Dr. Powell's paper they had been using successfully what they had come to know as the "navel sign"; if something attractive, such as a coin, was placed on the child's umbilicus while the child was at rest in bed, it would start to look at it by raising the head, but would be unable to do so in acute poliomyelitis. The performance of lumbar puncture had become a last resort rather than an early one, but it was well to remember that even if the cell count was normal the patient might have poliomyelitis and that the presence of more than forty lymphocytes per cubic millimetre in a suspicious case was a probable finding in acute poliomyelitis.

DR. A. P. DERHAM said that in his experience as the health officer of the city of Kew, he had found that "nose clips" and masks were probably of use in an infected household, and they had some educative value as they stressed the idea of the avoidance of breathing large masses of virus organisms. He conceded that as a general measure of prevention the clips were of little or no use in an industrial suburb like Carlton; but he had personally satisfied himself that with the clip on the outside of the nose he could not smell through the mouth, and he believed that with the return of the sense of smell protection ceased. Between two and three thousand of the clips had been issued to members of the public in Kew. In no community would they be used consistently or intelligently; but by their use it was possible to minimize the risk in contact nursing of the sick child. He had found that people were willing to wear face masks and nose clips in the house, though they would not wear them in the street; and he advocated that doctors should wear these protections when in contact, if not as a measure of protection, at least as an example to other people. Dr. Derham, too, regarded Dr. Powell's paper as a masterpiece. He said that in the early stage of poliomyelitis a child who was feverish and drowsy when not disturbed, became roused and cooperative when approached, and after the doctor's examination was over became drowsy again.

DR. J. B. COLQUHOUN referred to a patient who was noticed in July, 1937, to have wasting of the deltoid and upper arm muscles and was regarded as suffering from poliomyelitis, but it was found that the surgical neck of the humerus was fractured. In the after-care work at Caulfield he regarded the wearing of gowns and masks as a useful measure of discipline for nurses. He had met Sister Kenny and had been much impressed with her amazing personality as an individual, and was satisfied that in Queensland her work had resulted in general improvement in the after-care of patients with poliomyelitis. He had heard that the commission in New South Wales which was reporting on Sister Kenny's methods had reached the conclusion that while in her care patients with poliomyelitis improved, but not more rapidly than would be expected if in the care of orthopædic surgeons; that the results had been encouraging in spastic paralysis; and that her methods did not replace, but supplemented, the usual medical after-care of the patients. When she stated that she did not believe that paralysed muscles required rest and that the atrophy was caused by the splinting, she showed that she had very little idea as to what she was doing, and tragedy might result. She induced the patients to concentrate their attention on the space between the attachments of the muscles that were being actively exercised, and she showed her good sense by doing her work in pools, thus getting the aid of hydrotherapy, and by insisting on minimal contractions of the muscles early in the piece. Dr. Colquhoun pointed out that in reeducation it had to be remembered that the action of the opposing muscle lessened the chance of the recovery of the paralysed muscle and that deformities should be prevented. The important muscles were those that were later in the development of function, such as the deltoid and gluteal muscles, and that fatigue must be avoided in paralysed muscle tissue.

DR. J. P. MAJOR, from the chair, expressed thanks on behalf of those present to Dr. Scholes for his address that evening.[1]

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Cussen, Kevin Richard, M.B., B.S., 1930 (Univ. Melbourne), F.R.C.S., c.o. M. L. Cussen, Esq., Law Courts, Melbourne, C.1.

[1] The first part of the meeting at Warragul took the form of a series of clinical demonstrations; these will be reported in a subsequent issue.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on Wednesday, July 14, 1937, at the Children's Hospital, Carlton, Melbourne, DR. ROBERT SOUTHBY, the President, in the chair. The meeting took the form of a number of clinical demonstrations followed by a discussion by the members of the Society.

Exophthalmic Goitre.

DR. J. S. SWEETING presented for DR. STEWART FERGUSON a boy, aged eleven years, who had been admitted to the hospital on May 29, 1937. The patient's birth weight was 3·4 kilograms (seven and one-half pounds); he had been breast fed for three months, but had failed to thrive, and the feedings had been complemented with "Lactogen" and later with cow's milk mixtures. The boy's health had not improved, and medical advice was sought; he was found to have a congenital heart lesion. He had always been small and underweight, and though at times he appeared to be hungry, he ate little and picked at his food. His pulse rate was observed to be unusually rapid. The patient's only definite illnesses had been measles and mumps, and the family history showed nothing of importance.

Six months prior to the present illness, it had been noticed that he became distressed in his sleep, with rapid breathing and rapid pulse. Nothing relieved this, but he preferred two pillows. Medical advice was again sought, and he was given 0·032 gramme (half a grain) of thyreoid extract three times a day. Treatment had been continued for three months and had then been stopped, because it made the pulse more rapid; and he had improved slightly since it was stopped. One month later it was noticed that the eyes were protruding; this appeared to come on fairly suddenly, and had not since noticeably increased. Two months later a swelling in the neck was noticed. The child had always been very active, rather restless and unable to sit still for very long. The patient's weight had been 19 kilograms (three stone) for twelve months; he slept with the usual number of bedclothes and did not sweat much.

On examination, he appeared undersized, with small and pointed features, and pronounced bilateral exophthalmos; he was restless and had twitching movements of the mouth and hands. The apex beat of the heart was forcible and diffuse, and was localized about 2·5 centimetres (one inch) outside the usual position, and a systolic thrill was felt to the left of the sternum. There was a forcible first sound at the apex, with triple rhythm and a systolic bruit all over the precordium, which was more audible at the base of the heart. The systolic blood pressure was 160 and the diastolic blood pressure was 60 millimetres of mercury. There was a smooth, diffuse swelling of the thyreoid gland visible and palpable in the neck; a bruit was present on auscultation. There was one finger's breadth of liver dulness below the costal margin. The left testis was incompletely descended, and could be felt in the inguinal region. The skin was dry except for the soles of the feet and the palms of the hands, which were moist and clammy; there were coarse tremors in the hands. Pronounced exophthalmos of the eyes was noticeable. It was found that the pulse when the child was sleeping remained fairly constantly round 100 beats per minute. The basal metabolic rate was +52% on June 8, 1937, and +28% on June 12, 1937. A skiagram of the chest reveal prominence of the pulmonary artery on the left side, and right- and left-sided heart enlargement. There was no reaction to the Wassermann test, and an electrocardiogram revealed early right-sided preponderance. After the investigations the patient was treated with a course of Lugol's iodine solution and bromide. An interesting feature of the case was the patient's diet. Before admission he had been very difficult to feed, eating mainly carbohydrate and starchy foods, biscuits and honey being his particular favourites. He had, at the time of the meeting, however, with some trouble, been coaxed to take a good, well-balanced diet, and was apparently enjoying all that was offered to him. While he had been in hospital it was considered that the exophthalmos had become a little less prominent.

DR. STEWART FERGUSON said that he had thought that it would be of interest to the members to see the boy, because of the comparative rarity of the condition. He and Dr. Sweeting had felt that the boy was improving under medical treatment, and in addition to the diminution in the amount of exophthalmos, the basal metabolic rate had fallen and so had the pulse rate. He would like to hear a discussion on the most suitable method of treatment, and on the experiences that other members had had with the management of these patients. He had seen only three such patients, the youngest of whom had been two and one-half years of age. He remembered particularly a girl, aged six years and eleven months, concerning whose treatment he had consulted with the late Sir Richard Stawell, who had given an opinion against operative treatment. After medical treatment for eight months, during which the pulse rate increased and the heart became dilated, Dr. Julian Smith had performed a two-stage operation. The operation had taken place in 1928, and had been successful. The pulse rate had fallen to seventy beats per minute and the size and action of the heart had returned to normal. Dr. Ferguson had ascertained that the child had remained perfectly healthy since then. He felt inclined to advise operation in the case of the boy who had been shown that night. It was a difficult question to decide how much of the thyreoid gland should be removed and how much should be left in the case of a growing child. In addition, it had to be remembered that the child had improved under medical treatment. He was doubtful about the benefit to be obtained by means of X ray treatment, and he understood that if the doses used did not suit the case the outcome might be disastrous.

DR. JOHN F. WILLIAMS said that he thought that a psychological examination was indicated. There had been a sudden onset of severe symptoms and signs, and the child was fidgety and indulged in nail-biting. Before anything surgical was undertaken, the family surroundings and the child's psychological history should be investigated, in the hope that some factor would be brought to light, the removal of which might influence the condition favourably. It was well recognized that such stresses could bring on hyperthyreoid attacks.

DR. V. L. COLLINS said that he had communicated with the seven patients who had been admitted to the hospital since 1920 suffering from exophthalmic goitre. Three had replied, and had come to see him at his request. A girl, aged twenty years, had been in hospital in 1930 with gross exophthalmos and thyreoid enlargement; she had had chorea at the same time. Lugol's solution had been administered for a very short time and had been continued in a dose of two minims once a day, together with a little tincture of digitalis, for a while after her discharge from hospital. She had remained quite well; when he saw her he had noted the presence of slight exophthalmos, but the thyreoid gland was of normal size. Evidence of the presence of cardiac lesions ascribable to rheumatic heart disease had been obtained in addition. Another of these patients was a boy who underwent treatment for *diabetes mellitus* in the hospital in the care of Dr. M. Kent Hughes. He had contracted exophthalmic goitre in addition. He had required sixty units of insulin daily and still needed the same amount; but the appearance of the eyes had returned to normal. He had commented that, whereas people used to be rude to him about his eyes, no one noticed anything unusual at the time of the meeting. The thyreoid gland was also normal in size. He had continued to take iodine for a short period after his discharge from hospital. The third patient was a boy, who had been in the hospital the previous year. He was gaining weight, and the exophthalmos had decreased. He no longer took iodine, but was still receiving general treatment.

DR. J. W. GRIEVE said that he remembered all the children to whom Dr. Collins had referred, and he had been struck by the fact that, in his experience, exophthalmic goitre in children would subside with ordinary medical treatment.

DR. F. KINGSLEY NORRIS said that he remembered seeing in consultation a small boy with congenital heart disease and a history of constipation, and he had recommended the administration of thyreoid gland tablets. Recently he had seen the child again, and found him to have flagrant exophthalmos. He had wondered whether the thyreoid therapy had been a causative factor. That night he had seen the patient again; he was the child who had been presented that evening. At the last consultation he had expressed the opinion that it was quite possible that the boy would require an operation. He wondered whether exophthalmic goitre could be produced by treatment with thyreoid gland if the patient's thyreoid gland was normal. Dr. Norris, in view of the progress of the patient since he had seen him previously, believed that he was going to settle down under medical treatment. He did not like the idea of X ray therapy if it was possible that an operation would be performed at a later date.

DR. J. W. WHITAKER said that he had under his care a girl, aged thirteen years, with acute exophthalmic goitre. He had been very interested in Dr. Grieve's remarks on prognosis. He had withheld operation because, though the girl was of mature appearance, she had not menstruated. He considered that there was a mixed endocrinal disturbance in her case, with some ovarian upset as well as the functional thyreoid abnormality. He had not had any experience of the surgical results in this disease.

DR. WILFRED FORSTER said that he had seen three children suffering from exophthalmic goitre, all of whom were girls. Two were twelve years of age, and in each case the disorder had disappeared at the onset of menstruation after a little iodine treatment. In the third case, the thyreoid gland had been large, and Dr. Forster had performed bilateral thyreoidectomy. Four days after the operation the girl had shown evidence of paralysis of the recurrent laryngeal nerve. Apart from the persistent hoarseness, the result had been gratifying.

Dr. Stewart Ferguson thanked those who had contributed to the discussion, and said that he had been particularly impressed by the opinion against operation expressed by Dr. Grieve. Nevertheless, he was able to quote another case of a girl, aged seven years, who had not obtained any apparent benefit from medical treatment over a period of nine months and had responded immediately to surgical treatment. He thought that he would soon be asking a surgeon to consider operation on the boy shown that night.

Amaurotic Familial Idiocy.

DR. J. W. GRIEVE showed a female, aged one year and three months, with amaurotic familial idiocy. The patient was a full-time, normal baby, the birth weight being 3·77 kilograms (eight pounds five ounces). The mother had normal labour without any instruments, and the child had given no evidence of cranial injury after birth. She had been breast fed for six weeks, and during that time had developed and gained weight satisfactorily. The patient's mother noticed that she did not hold her head up at the usual age, that her limbs were very flabby, and that she moved very little. The child had remained in the same condition, unable to hold her head up or sit up and having very little interest in her surroundings, particularly with regard to visual sensations. The immediate cause of admission to hospital was an attack of convulsions. There were no other children in the family, and there had been no miscarriages or stillbirths; both the father and mother were healthy; there was no family history of insanity or blindness. On examination, the child appeared well nourished and large for her age; the head was large and misshapen, and she had a vacant expression on her face. The heart and abdomen were clear, but she had some bronchitis. The deep muscle reflexes were normal; the plantar reflexes were equivocal and the superficial abdominal reflexes were doubtful. There was no neck stiffness or Kernig's sign. There was generalized extreme hypotonia, but the limbs were not wasted. The pupils both reacted to light, but the right pupil was slightly larger than the left. There was a cherry-red spot at each *macula lutea*, with a surrounding pale halo. There was optic atrophy of both eyes, more pronounced in the right.

Dr. Grieve commented on the rarity of this condition, particularly in a family which was not Jewish and had no history of Jewish ancestry. He had noticed a report of a series of 200 cases of this cerebro-retinal degenerative condition, in which eighteen of the children were not Jewish. At first he had thought that the child was an example of the more common cerebral sclerosis, until Dr. J. S. Sweeting had discovered the cherry-red spot at the macula. The eyes moved rapidly and the changes were difficult to see, but he and Dr. Gardner had confirmed their presence.

DR. GRAEME ROBERTSON said that the infantile form of amaurotic familial idiocy was associated with the names of Waren Tay and Sachs, and was usually diagnosed by the time the baby had reached the age of eight months. The child wasted away gradually, frequently had convulsions and invariably died, generally within six months. The condition was a specific ganglion cell disease, and could be classified with the disorders of lipoid metabolism. It was in some way associated with Niemann-Pick's disease, with Schüller-Christian's disease, and with Gaucher's disease. Dr. Robertson illustrated his remarks with a number of lantern slides.

Chronic Toxæmia.

DR. M. A. MACKIE, on behalf of DR. A. P. DERHAM, showed a little girl, aged four years, who had been admitted to the Children's Hospital two months earlier after having been in a country hospital for a year on account of a long febrile illness. When originally admitted to the country hospital, she had had a high, swinging temperature, and pus and albumin had been present in the urine. Lumbar puncture was performed because of meningismus, but the cerebro-spinal fluid was normal. Two weeks later the child had contracted gastro-enteritis, and had continued with diarrhœa for about five weeks. At that stage she had had an attack of pneumonia, after which the urine had become clear. For a further eight months the child had periods of pyrexia, the temperature reaching 40·55° C. (105° F.) at times, associated with enlargement of the glands of the neck, groin and axillæ, and with protuberance of the abdomen at times. Towards the end of her stay at the country hospital the child had had another attack of diarrhœa, with offensive, fatty stools, and the spleen and liver had become palpable; a discharge had occurred from each ear, and several of the fingers had become infected.

When the child was admitted to the Children's Hospital two months prior to the meeting, generalized enlargement of the glands and considerable enlargement of the liver and spleen were still present. The child had a cleft soft palate and was unable to talk. The temperature had run a normal course for two weeks, but for nine days the child's temperature seldom fell below 37·8° C. (100° F.), and there was a daily swing to 39·45° C. or 40° C. (103° or 104° F.). With this febrile interlude the glands became larger and the child was miserable. Evidence of diffuse bronchitis was obtained later. On May 19, 1936, the serum gave no reaction to the Widal test, and on seven occasions examination of the sputum had revealed no tubercle bacilli. Repeated attempts at blood culture had been made, but no organisms were grown. Radiographic examination of the chest had indicated that the heart was enlarged and that there was some suspicious mottling of the upper zones of each lung. On July 3, 1936, a moderate degree of secondary anæmia was present; the differential leucocyte count was normal. On April 19, 1937, however, the erythrocytes numbered 5,500,000 and the leucocytes 19,500 per cubic millimetre of blood, and the hæmoglobin value was estimated to be 80%; the differential count and the film appeared to be normal. On

January 26, 1937, a section of a lymph gland was prepared; it was seen by Dr. E. J. S. King, who commented on the similarity to the appearance of a gland from a patient with chronic staphylococcal infection. No reaction was obtained to the Mantoux and Casoni tests, and the urine was normal. The patient's serum had failed in the agglutination test for *Brucella abortus*. Blood sedimentation tests had been made on August 24, 1936, and June 24, 1937; on the earlier occasion the fall in millimetres in one hour was 13·5, and in June, 1937, during an apyrexial period, the fall was 18 millimetres in one hour.

The patient was shown because of the diagnostic difficulties, but no comment was forthcoming.

Tuberculosis of the Hip with Ureteral Calculi.

DR. J. G. WHITAKER showed a boy, aged sixteen years, who had been undergoing treatment for a period of three years at the Frankston Orthopædic Branch for tuberculous disease of the hip joint. Dr. Whitaker said that a more or less characteristic result had been obtained; there was some shortening and fair ankylosis, and the boy had been walking about in a plaster spica bandage. The point of interest in this case had been the development of multiple ureteral calculi. He was the only one of sixty-three patients who had developed a calculus of the ureter. Some writers had stated that calculi were more common at this site than in the kidney when associated with tuberculous disease of the hip joint. Dr. Whitaker showed a series of skiagrams in which it could be seen that a calculus was present in the left ureter at a comparatively early stage. He had removed this calculus at operation. At a later date it was ascertained radiographically that there was another calculus in the right ureter. He had been unable to get the boy to pass this small calculus, and it had increased in size. A "Uroselectan" investigation revealed nothing on the side on which the stone was present; the urea in the blood and in the urine on concentration was within normal limits. He would like to know how long one could leave the kidney functionless without damaging it permanently. Because of the renal suppression and hibernation, he had attempted to remove the stone from the right side. The kidney had dilated and hydronephrosis was probably present. He had exposed the stone and grasped it in his fingers, but it had disappeared and he could not find it any more. It must have disintegrated and come out in small pieces; it had been reported to him that the boy had passed gravel in the urine. The "Uroselectan" investigation had resulted in the reappearance of the kidney shadow. The absence of any kidney shadow previously could have been due only to dilution of the dye or to failure of the kidney to secrete, and he had taken the latter view.

Dr. Whitaker said that it would be of interest to members to know that there had been only one example of undoubted renal tuberculosis among the patients with tuberculous hip disease at Frankston, though tubercle bacilli had been obtained in the urine of three out of forty patients in whom repeated searches had been made.

DR. KEITH HALLAM discussed the radiography of calculous anuria in connexion with the patient shown by Dr. Whitaker. Dr. Hallam said that he had noticed that with excretion urography in the patients he had seen the kidney shadow became denser and denser over a period of thirty minutes. The "Uroselectan B" was excreted by the glomeruli into the tubules, but it did not reach the calyces or the pelvis. He attributed this finding to a reflex spasm in the main collecting tubules, excited by the presence of a calculus in the pelvis or ureter. He explained that the distinction between hibernation and calculous anuria was of great importance; in the former condition there was complete destruction of kidney function, which amounted to autonephrectomy at times. If either condition was temporary, the renal function could return to normal. Calculous anuria could be diagnosed when the kidney substance was seen to be growing denser and denser with secretion, but when no excretion of dye took place.

Dr. Whitaker repeated that in the patient he had shown no kidney shadow whatever had appeared.

Fractured Femur.

Dr. Whitaker also showed a child who had been treated for a fractured femur in a modification of Braun's splint, which had first been shown to Dr. Whitaker by Dr. Edward Prendergast. By means of a system of pulleys it was possible to get flexion at the hip and knee when required, and also to overcome the tendency to backward displacement of the femur, which was a difficulty encountered in the ordinary Braun's splint. The child was nursed on a small bed with a divided mattress, and a block was fixed at the end of the bed for pressure on the sound side. The child was thus able to stabilize herself on the splint and to be nursed satisfactorily.

DR. WILFRED FORSTER expressed emphatically his dissatisfaction with the ordinary Braun's splint.

(To be continued.)

Correspondence.

COMMON PROBLEMS IN GENERAL PRACTICE.

SIR: Dr. Winter Ashton, quite inadvertently I am sure, in his reply to my letter, misstates my main proposition. It was he, not I, who complained of the lack of representation of the general practitioners on the Council of the Victorian Branch of the British Medical Association. He pointed out quite truly that this is the fault of the general practitioners themselves.

My contention is that the council, however constituted, should not purport to voice the feeling of the profession on important matters without being sure that it knows what that feeling is. The presence of a few extra general practitioners on the council would not affect this principle one iota.

Our profession has been described, not inaptly—I forget by whom—as one "which keeps its mouth shut and confines its writing to prescriptions". It may be, therefore, often impossible to ascertain the general feeling of the profession without going to some trouble, such as sending out a *questionnaire*.

In the case in point—a proposal to seek legislation to abrogate most of the present restrictions on abortion—no such inquiry has been made. I venture to think that, if it should be made, the result would differ from that apparently desired by Dr. Ashton.

Yours, etc.,

H. G. LOUGHRAN.

Sari Bair,
Kyneton,
Victoria,
November 26, 1937.

TUBERCULOUS AND BACILLUS COLI COMMUNIS INFECTIONS.

SIR: I have been treating cases of *Bacillus coli communis* infection for over twenty-five years, and I have been puzzled at the remarkable persistence of the trouble in spite of up-to-date treatment, and to find that for some unknown reason it will disappear and the patient remain free in some cases for more than twenty years. If there is a complaint that a quack can claim success in treating it is this one. But he has more failures than successes because the trouble recurs.

I am of opinion that the *Bacillus coli communis* infection is the result of an association of the organism with that of tubercle and that it is the ultravirus of this latter which

is the real agent. When this clears up the *Bacillus coli communis* goes with it.

Now tuberculous infections can and do remain silent for years and then become active for longer or shorter periods and go to sleep again.

We can desensitize the patient with vaccines against the toxins of *Bacillus coli communis*. Alkalis, according to the late Gaston Billard, probably act by combining the lipoids of the cells, forming soaps and preventing the toxins of the *Bacillus coli communis* from fixing on the cells and damaging them, consequently clearing the urine in urinary forms. Mandelic acid, hexamine *et cetera* probably block the toxins from becoming fixed.

But until the tuberculous activity ceases the patient will not get better, and his recovery will be expedited by the return of hot weather.

Yours, etc.,

J. MORRIS ROE.

Victory Chambers,
Queen Street,
Brisbane,
November 29, 1937.

TUBERCULOSIS.

SIR: Dr. J. G. Edwards takes me to task in THE MEDICAL JOURNAL OF AUSTRALIA of November 20 for my advocacy of fluoroscopy in the diagnosis of pulmonary tuberculosis. I would point out that the statement to which he takes exception occurs in a paragraph in my paper in which I emphasize the need for periodic health examinations of selected groups of young adults, apparently in health, in order to discover pre-clinical cases of tuberculosis. To carry out such a survey, X ray examination *en masse* is essential. The first question that arises is that of practicability. If Dr. Edwards is correct in his contention, the problem would be one of mass radiography, the cost of which in this country would probably be prohibitive.

The next question that will be asked is: does fluoroscopy give adequate results? From my reading of the literature and from personal experience in screening patients who are undergoing artificial pneumothorax therapy I am convinced that this question can be answered in the affirmative. Fluoroscopy will reveal processes of quite small extent; but if such are observed, a check radiograph must also be taken. Even this, of course, in some cases cannot determine with certainty whether a pulmonary infiltration is tuberculous. Much of the reluctance in the past to acknowledge fluoroscopy as an independent method of examination was due to lack of patience in ocular adaptation and to the use of the old type of fluoroscopic screen.

Fluoroscopy is now being employed in industrial and other surveys in America and Europe, and it is very desirable that we endeavour in Australia to fall into line and to carry out these group surveys. No one will pretend that a good film is not of greater value than fluoroscopy in any individual case, and I am in accord with Dr. Edwards in not giving an opinion in a doubtful case on fluoroscopy alone. The practical point, however, on which I would insist, is that fluoroscopy in chest work is reliable as a means of detecting the presence of pathological processes, and it is far less costly than radiography. It is excellently adapted for carrying out large scale examinations in factories, schools and the like, and it will prove to be a very important factor in the future campaign against tuberculosis.

Dr. Edwards's remarks on the dangers of fluoroscopy do not apply. The examinations would not be frequent—for the patient there would be no "repeated fluoroscopy"—and would be of comparatively short duration, and the physician would naturally be adequately protected against irradiation.

Yours, etc.,

COTTER HARVEY.

British Medical Association House,
137, Macquarie Street,
Sydney,
December 3, 1937.

The Royal Australasian College of Surgeons.

MEETING OF THE BOARD OF CENSORS.

THE next meeting of the Australian Board of Censors of the Royal Australasian College of Surgeons will be held at the College, Spring Street, Melbourne, in March, 1938. Candidates who desire to present themselves at this meeting should apply to the Censor-in-Chief for permission to do so on or before December 31, 1937. The appropriate forms are available at the College and at the offices of the various State secretaries.

Notice.

PROPOSED AUSTRALIAN BRANCH OF THE DIABETIC ASSOCIATION.

A MEETING to consider the proposal to form an Australian branch of the Diabetic Association will be held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on Friday, December 17, at 4.30 p.m. All medical men, their diabetic patients and anyone interested in the social welfare of diabetics are invited to attend. The Honourable H. P. FitzSimons, Minister for Health, and other prominent citizens will be present.

THEFT OF SHORT WAVE THERAPY OUTFIT.

THE Police Department has requested us to make public the theft from a motor car at Waverley on the night of December 4 of an electric "Thermox" short wave therapy outfit. The machine was manufactured by P. A. Bowker and Company, and bears the identification mark "Westinghouse WL 460 valve" and the serial numbers 3034 and 303 U.

Any medical practitioner to whom such an outfit should be offered for sale, or who should chance to come in contact with it in any way, is asked to communicate at once with the local police.

Obituary.

THOMAS GEORGE BECKETT.

WE regret to announce the death of Dr. Thomas George Beckett, which occurred on November 22, 1937, at Melbourne, Victoria.

JOHN WILLIAM O'BRIEN.

WE regret to announce the death of Dr. John William O'Brien, which occurred on November 26, 1937, at Toorak, Victoria.

ROBERT JAMES ORFORD.

WE regret to announce the death of Dr. Robert James Orford, which occurred on December 5, 1937, at Green Point, Gosford, New South Wales.

Books Received.

BIOLOGY FOR MEDICAL STUDENTS, by C. C. Hentschel, M.Sc., and W. R. I. Cook, B.Sc., Ph.D., with a foreword by G. E. Gask, C.M.G., D.S.O., F.R.C.S.; 1937. London: Longmans, Green and Company. Demy 8vo, pp. 676, with illustrations. Price: 18s. net.

MEDICAL RESEARCH COUNCIL OF THE PRIVY COUNCIL. SPECIAL REPORT SERIES, NUMBER 224. AN ANALYSIS OF THE RESULTS OF TREATMENT OF EARLY, LATENT, AND MUCO-CUTANEOUS TERTIARY SYPHILIS, by W. R. Snodgrass and R. J. Peters; 1937. London: His Majesty's Stationery Office. Medium 8vo, pp. 140. Price: 2s. 6d. net.

MEDICINE: ESSENTIALS FOR PRACTITIONERS AND STUDENTS, by G. E. Beaumont, M.A., D.M., F.R.C.P.; D.P.H.; Third Edition; 1937. London: J. and A. Churchill Limited. Medium 8vo, pp. 798, with 74 illustrations. Price: 21s. net.

COLLECTED PAPERS OF THE BAKER INSTITUTE OF MEDICAL RESEARCH, ALFRED HOSPITAL, MELBOURNE. NUMBER III, CONTAINING 55 PAPERS; 1937. Melbourne: The Baker Medical Research Institute. Crown 4to, with illustrations.

Medical Appointments.

Dr. L. E. Hurley has been appointed a Certifying Medical Practitioner at Melbourne, in accordance with the provisions of the *Workers' Compensation Acts*.

* * *

Dr. W. V. Tothill has been appointed Medical Officer of Health to the Wagin Local Board of Health, Western Australia.

* * *

Dr. A. B. Barry has been appointed Deputy Medical Superintendent in the Department of Mental Hospitals of New South Wales.

* * *

Dr. H. N. Merrington has been appointed Medical Referee for the purposes of *The Workers' Compensation Acts*, 1916 to 1936, of Queensland.

* * *

Dr. C. A. Finlayson and Dr. J. S. Proctor have been appointed Official Visitors to the Mental Hospital at Parkside, South Australia, pursuant to the provisions of the *Mental Defectives Act*, 1935, of South Australia.

Diary for the Month.

DEC. 14.—Tasmanian Branch, B.M.A.: Branch.
DEC. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 15.—Western Australian Branch, B.M.A.: Branch.
DEC. 17.—Queensland Branch, B.M.A.: Council.
DEC. 21.—Tasmanian Branch, B.M.A.: Council.
DEC. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 30.—South Australian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xx to xxii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Resident Medical Officer.
MARYBOROUGH HOSPITALS BOARD, MARYBOROUGH, QUEENSLAND: Junior Resident Medical Officer.
MOUNT MAGNET HOSPITAL, MOUNT MAGNET, WESTERN AUSTRALIA: Medical Officer.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Medical Superintendent.
THE TOOWOOMBA HOSPITALS BOARD, TOOWOOMBA, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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